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Volume 14 No. 4
October • 1959

THE CENTENNIAL YEAR — 1959

ARTICULOS SUMMARISATE
IN INTERLINGUA

journal of dental medicine

OBJECTIVES OF THE AMERICAN ACADEMY OF DENTAL MEDICINE

(Quoted from the Constitution)

"To promote the study and dissemination of knowledge of the cause, prevention and control of diseases of the teeth, their supporting structures and adnexa and related subjects; and to foster and promote a better scientific understanding between the fields of dentistry and medicine."

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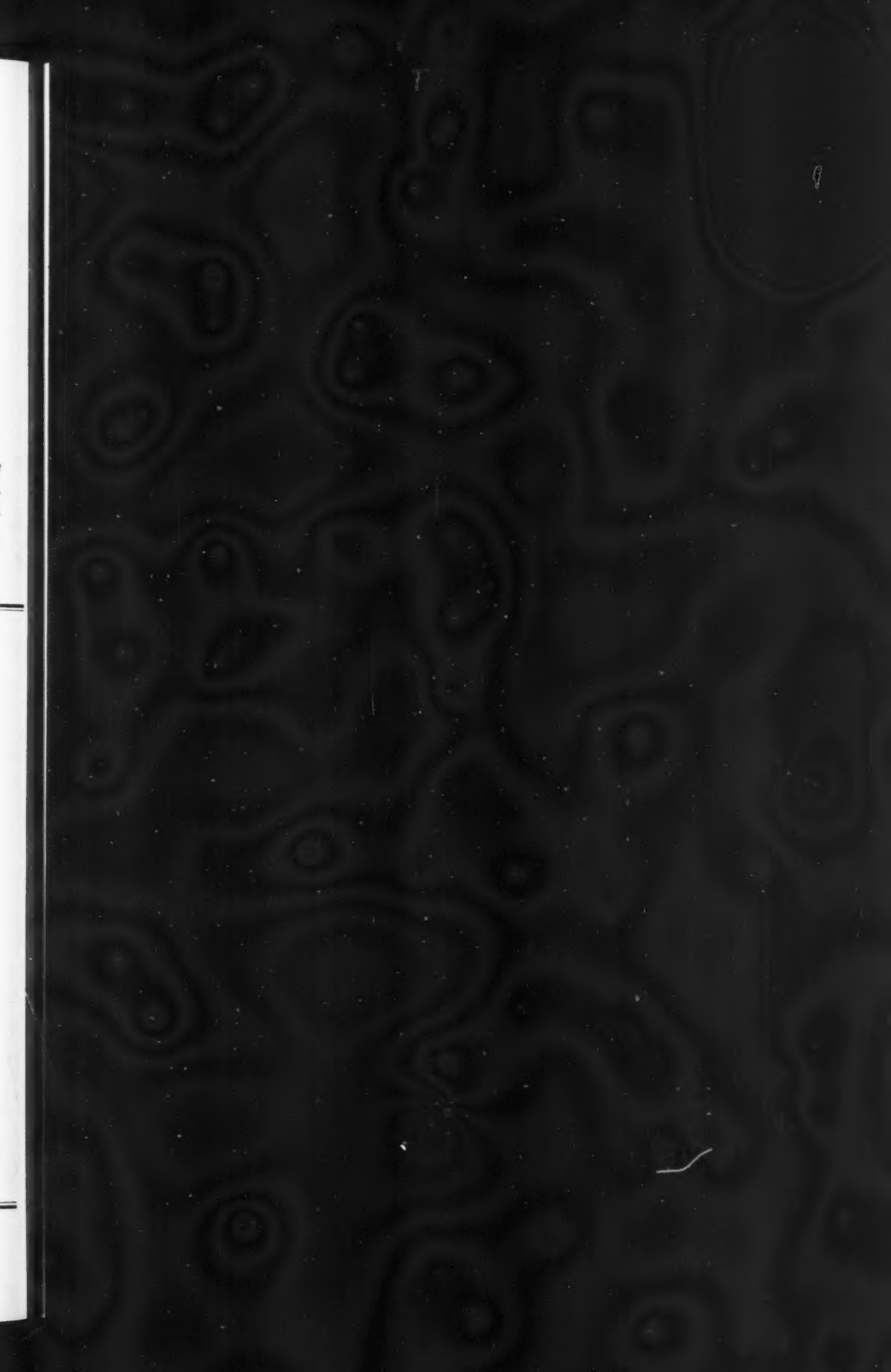

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JOURNAL OF DENTAL MEDICINE

VOL. 14 OCTOBER, 1959 NO. 4

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Subscription rate: $6.00 Annually, $6.50 Foreign. Single copies, $1.50. Special Nutrition Issue (Sept. 1953) $1.00 per copy, special rate in quantity. Tenth Anniversary Issue (April 1956) $1.00 per copy, $5.00 for bound volume. Special student subscription rate, $4.00 annually.

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2. The title should appear at the top of the first page, followed by the name and degrees of the author or authors. The address of the author should appear at the end of the article.

3. The name of the group before which the paper was presented, the date and place of delivery and the author's university, hospital or government affiliation, if any, should appear at the bottom of the title page.

4. Manuscripts presented for publication should: a—present new facts, describe new techniques or methods of practice or reevaluate established principles; b—contain the results of original research, or c—provide a review of facts concerning a particular subject so that the reader may deduce therefrom legitimate, important conclusions.

5. Data presented in charts and tables should be discussed but not repeated in the text.

6. A concise summary and a precise conclusion add to the value of a scientific article. The summary should state briefly the purpose of the article and the main facts presented or reviewed.

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A Salute to the American Dental Association

The American Academy of Dental Medicine is happy to participate in celebrating the 100th anniversary of organized professional dentistry in this country. We pay tribute to the wisdom and foresight of the founders of the American Dental Association, and to all of the men and women, who have contributed their energies, as leaders of this great organization. As a result of their efforts, Dentistry has gained stature, as a profession, and the American people have received improved health services.

In this second century of professional growth our aim should be the continued improvement of the level of oral and general health of the total man in his total environment. We should also make every effort to share our achievements with all of our neighbors and friends, throughout the world.

These are the aims of the American Academy of Dental Medicine. The scientific and social program of this Centennial Meeting initiates that goal for organized American Dentistry.

GEORGE G. STEWART, *President*

SALUTE AL ASSOCIATION DENTAL AMERICAN

Le Academia American de Medicina Dental es felice de participar in le celebration del centesime anniversario del organisate dentisteria professional in iste pais. Nos paga tributo al sagessa e al perspicacitate del fundtores del Association Dental American e a omne le homines e feminas qui ha contribuite lor energias como ductores de ille grande organisation. Como resultato de lor effortios, le dentisteria ha crescite in corpore e spirito como profession, e le populo american ha gaudite de meliorate servicios de sanitate.

In le veniente secunde seculo de crescentia professional nostre objectivo debe esser le continuate melioration del nivellos de sanitate dental e general del parte del individuo total in su ambiente total. Nos etiam debe facer omne effortio de compartir nostre attingimentos con omne nostre vicinos e amicos in omne partes del mundo.

Istos es le objectivos del Academia American de Medicina Dental, e le programma scientific e social de iste Assemblea Centennial initia ille curso pro le organisate Dentisteria American.

GEORGE G. STEWART, *Presidente*

HIGHLIGHTS IN THE HISTORY OF DENTISTRY 1859-1959

1859	The American Dental Association was founded.
c. 1860	Vulcanite came into general use as a denture base.
1867	The first university dental school was founded at Harvard University.
1868	Effective licensure legislation was instituted by Kentucky, New York and Ohio.

1880-1890 W. D. Miller's *researches* established the basis for the present theory of the etiology of caries.

c. 1905 Procaine was introduced into dental practice.

1906 William H. Taggart introduced a practicable method of pressure casting of gold that revolutionized restoration of teeth and crown and bridge work.

1907 Angle's 7th edition of his *Malocclusion of the Teeth* systematized the treatment of malocclusions.

1910 Dentists became much concerned about the relation of diseased teeth to disease elsewhere in the body (focal infection).

c. 1910 X-ray began to be employed as a means of diagnosing dental disease.

1911-1912 Dental officers were commissioned in the Armed Forces.

1913 *The Journal of the American Dental Association* was established under the title *Bulletin of the National Dental Association.*

1921 The Association for Dental Research was established.

1926 The Carnegie Report (by William Gies) on Dental Education in the United States and Canada was issued.

1928 The National Bureau of Standards and the American Dental Association, in cooperation, began the standardization of dental materials.

1930 The Council on Dental Therapeutics was established to insure the quality and character of dental remedies.

c. 1937 Acrylic resin began to be employed as a denture base in place of vulcanite.

1938 The Council on Dental Education of the American Dental Association was established.

1944 Field trials began of fluoridation of water supplies as a measure for control of caries.

1948 National Institute of Dental Research was established at Washington, D. C.

1949 To the historians of the next 100 years must be left the task of cataloguing the activities and achievements of this past decade. We are still too deeply involved in these activities to evaluate clearly their lasting impact on the profession and on the public. However, among the highlights of this decade must certainly be counted such events as the start of the aptitude testing program for pre-dental students; the intensification of dental research as witnessed by the expanded activities of the Association's Council on Dental Research and the multi-million dollar expenditures for research studies by the National Institute of Dental Research; the increasing emphasis in dentistry on prevention of disease and on concern for the total health of the patient; the increasing use of auxiliary personnel by the dentist, and the growing demand for dental service by the public.

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RECENT PROGRESS IN STRESS RESEARCH*

HANS SELYE, M.D.†

Ever since the description, in 1936, of the general adaptation syndrome (the body's response to nonspecific stress),[1] numerous investigations were published throughout the world, in an attempt to clarify the mechanism through which, depending upon circumstances, stress can either produce or prevent disease.[2] More particularly it has been the major endeavor of our Institute to prove that hormones can play a decisive role in the development of several nonendocrine diseases, that is, of maladies which are not primarily due to derangements originating in the endocrine glands themselves.

During the last 20 years, it has been amply demonstrated that in response to a variety of stressors (trauma, infections, intoxications, nervous stimuli, etc.), the body reacts in a rather stereotyped manner by the secretion of certain hormones such as ACTH and corticoids. The resulting excess in these "adaptive hormones" is an essentially useful defense reaction. However, under certain circumstances, it can become the cause of disease or, at least, predispose the body to the production of morbid changes. For example, the excessive secretion of ACTH and of anti-inflammatory corticoids is useful because it helps the body to survive during an emergency, and because it suppresses excessive inflammatory reactions; but, at the same time, it can also be harmful in that it may enhance the spread of infections or predispose to the production of peptic ulcers.

It was my privilege—on two previous occasions—to present before the American Institute of Dental Medicine a detailed outline of our findings on stress.[3] The major results of our investigations were subsequently summarized in a monograph for the general public, *The Stress Of Life*,[4] so that I shall limit myself here to a discussion of one of the main aspects of our work over the past two years:

EXPERIMENTAL BONE DISEASES AS TOOLS FOR RESEARCH ON THE PHYSIOPATHOLOGY OF BONE WITH SPECIAL REFERENCE TO BONE HEALING

Osteolathyrism—Recently a monograph was published, the object of which was to bring together the numerous but scattered observations that deal with clinical and experimental lathyrism. Many of these findings appear to be quite unrelated to each other, but their systematized coordination was a necessary prerequisite to the rational analysis of this curious malady.[5]

Lathyrism in man is essentially a nervous disease caused by the ingestion of certain pulses, particularly Lathyrus sativus; it is unaccompanied by any significant changes in the skeletal system. A very similar type of "neurolathyrism" occurs in domestic animals (horses, cattle, etc.), whose feed consists predominantly of the same kind of pulse.

An apparently quite unrelated skeletal disease, "osteolathyrism," can be induced experimentally in laboratory animals by feeding them the seeds of other types of Lathyrus plants, especially L. odoratus. The active principle of the latter is aminopropionitrile (APN). This substance has been isolated from the seeds of L. odoratus and, in animals, it reproduces the characteristic skeletal lesions that occur after feeding the pulse itself. Several synthetic nitriles —which are not known to occur in food— share this osteolathyrogenic property of APN, but other related nitriles produce predominantly nervous manifestations, and there are some cognate compounds which induce typical skeletal changes in combination with such nervous derangements. Interestingly, a derivative of APN, (aminoacetonitrile (AAN), which is not

* Presented before the 15th Annual Meeting of The American Institute of Dental Medicine, Palm Springs, California, October, 1958.

† Institute of Experimental Medicine & Surgery, University of Montreal, Canada.

known to occur in nature, possesses an even greater osteolathyrogenic effect than APN.[6, 7]

The syndrome of osteolathyrism was not recognized until 1933, when Geiger et al.[8] observed that rats fed on diets rich in L. odoratus peas developed deformities in the long-bones, spinal curvature, distortions of the thoracic skeleton, and occasionally, hernias. Shortly afterwards, these observations were confirmed by Robinson and Bast[9] who also described in detail the histologic changes produced by the ingestion of L. odoratus in the rat. However, it has been quite justly pointed out[10] that the work of Ponseti and Baird[11] was of particular importance in calling attention to the possible clinical implications of experimental osteolathyrism. Ponseti and Shepard[12] expressed the view that the bone lesions in rats kept on L. odoratus diets reproduce many of the typical characteristics of various spontaneous skeletal diseases of man, for example: kyphoscoliosis, slipped epiphyses, herniation of intervertebral discs, degenerative arthritis, Legg-Perthes' disease, Paget's disease of the bones. They found, furthermore, that all the experimental lesions are frequently associated with inguinal hernias and dissecting aneurysms, which suggested that L. odoratus produces a systemic lesion of mesodermal tissues.[12, 13] This relationship to common clinical conditions was further emphasized by the finding that dissecting aneurysms of the thoracic aorta frequently occur in combination with kyphoscoliosis in human beings.[14]

The first clinical evidence of osteolathyrism is a peculiar "waddling" gait which usually becomes manifest after about one week of AAN treatment. At that time the skeletal structures appear to be very tender to the touch, and the rats usually lie on their sides or backs, evidently because of pain in their limbs.

Upon palpation, signs of bone-lathyrism are first detectable in the mandible and knee-joint regions. The muscle attachments on the mandible become prominent; the proximal metaphysis of the tibia is enlarged and bent backwards, while the adjacent end of the fibula protrudes under the skin, owing to its dislocation by the curved tibia.

A few days later the muscle attachments, especially along the inner surface of the femur and on the major trochanter, also become palpably enlarged and the shaft of the femur is greatly thickened. During the third and fourth weeks of treatment, obvious deformities develop in the spinal column, the thorax and the tarsophalangeal joints of the hind paws. There is kyphoscoliosis and the xiphoid bone is sharply bent near its middle and it protrudes forward in the form of a pointed hump. Then the distal ends of the metatarsal bones become greatly swollen (club-shaped) and, owing to this deformity, the digits deviate laterally. Finally, in very advanced bone-lathyrism, the tail is permanently curled up in a spiral fashion (often with several sharp kinks) and it cannot be straightened out by traction.

Sometimes the hind legs become paretic or completely paralyzed and reflex irritability in the hind quarters is greatly increased. This is evidently due to the compression of the spinal cord by the deformed spinal column.

The upper incisors often show a peculiar notch[15, 16, 17] near the tip (Fig. 1) and, under certain conditions, they may grow excessively so that eventually they turn backwards and up-

Fig. 1. *Aggravation of osteolathyrism by STH.* Incisor tooth of a rat treated with a small dose of AAN alone (left) and of one which received the same dose of AAN + STH. In the former rat, the teeth are essentially normal, while in the latter, there is marked malocclusion with several cross lines of enamel defects and excessive growth in length. The tips of the upper incisors have turned upwards and are growing into the palate and lips.

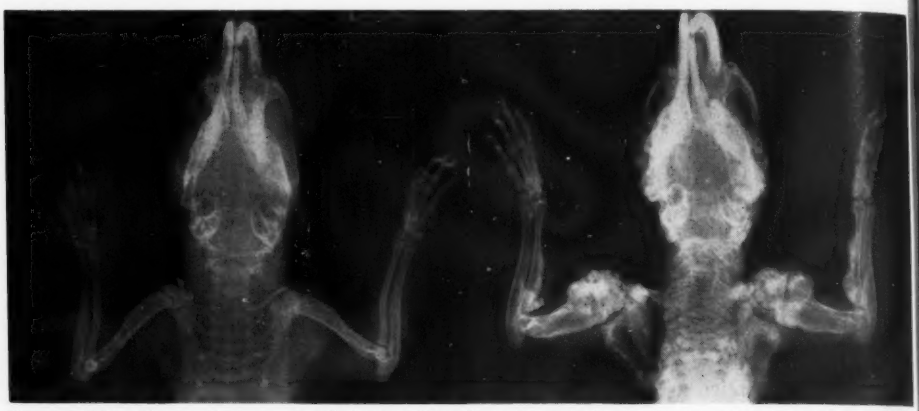

Fig. 2. *X-Ray picture of severe osteolathyrism.* Note bone irregularities around widened junction-cartilage plates, intense periosteal new bone formation, particularly at muscle-insertion sites (e.g., on mandibles), in comparison with a normal control rat.

wards towards the lips like tusks. Then, horizontal enamel defects appear on the anterior surface of the incisors (Fig. 1) and the teeth often break spontaneously.[18] These anomalies of the incisors are presumably due (at least in part) to the fact that the malformations of the jaw bones lead to malocclusion; consequently, the animal can no longer grind off its incisors (by chewing) as fast as they grow.

Finally, the skin becomes extremely thin, and the fur ruffled.

If much higher doses of AAN are administered, the syndrome is considerably more acute and there appear multiple hemorrhages, especially around muscle-insertion sites. These hematomas are clinically detectable, because the dark blood is visible through the thin skin.

In weanling rats, this acute osteolathyrism is induced with particular ease and often in combination with spontaneous fractures and dissecting aneurysms.

Apparently most of the common laboratory animals respond in essentially the same manner to lathyrogens. We elicited typical osteolathyrism in the cat, dog, guinea-pig, hamster, rabbit and monkey.[5]

In the rat the skeletal lesions are strikingly similar, irrespective of whether they are elicited by feeding the seeds of L. odoratus or by the enteral or parenteral administration of lathyrogenic nitriles (e.g., AAN, APN). These changes can be conveniently followed on X-ray pictures (Fig. 2). However, the character of these changes depends largely upon the age of the animals and the severity of the intoxication. Young rats are particularly sensitive, and tend to respond with a very acute syndrome which may develop within a week and which, in its severest form, terminates in death a few days after its appearance. The essential morphologic characteristics of this acute osteolathyrism are the following:

At autopsy, the bones are thickened, extremely fragile and soft; marked bone proliferation is visible, particularly at tendon-insertion sites throughout the skeleton, and multiple hematomas develop in the periosteum, particularly around the occiput and the mandibles. Spontaneous fractures occur most frequently in the ribs. The epiphyseal cartilages are greatly enlarged and partly liquefied; consequently, the epiphyses readily become detached from the diaphyses (Fig. 3).

It has been found, histologically, that bone formed under the influence of osteolathyrogens (e.g., AAN) is not normally canalized by the processes of osteocytes.

In the bone matrix the osteocyte canaliculi become fragmented as soon as they are formed and the severed parts become roundish "baso-

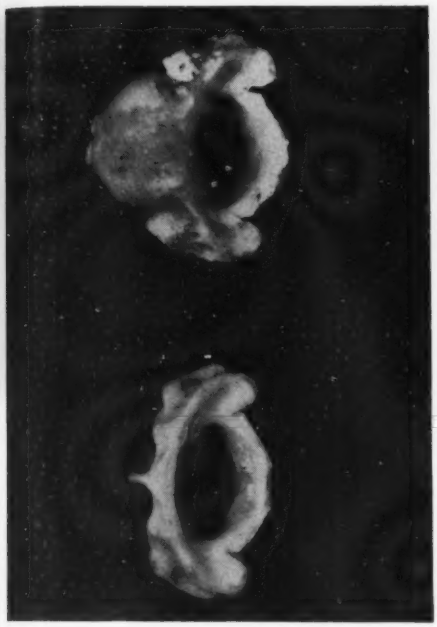

Fig. 3A

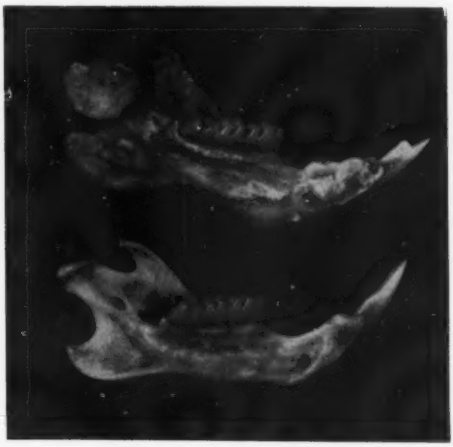

Fig. 3B

Fig. 3. *Inhibition of osteolathyrism by cortisol.—A.* Atlas (cranial view) following treatment with AAN (top) and AAN + cortisol. The excessive bone proliferation with the development of exostoses (most pronounced at the site of muscle insertions) is inhibited by cortisol. This inhibition is especially evident, as regards the enormous exostosis that always develops at the site of the anterior tubercule of the atlas. *B.* Left mandible (buccal view). Voluminous exostoses at muscle insertions—particularly obvious at the coronoid and condyloid processes and at the angle of the jaw—in a rat receiving AAN alone (top), while the bone structure remains essentially normal in the animal given AAN + cortisol. Note also abnormal configuration of the incisor tooth in the rat receiving no hormone treatment.

philic bone globules." A special stain (the celestine blue technique) has been developed for the selective staining of these corpuscules; another stain (the gentian violet technique) made it possible to visualize both the osteocyte canaliculi and the "basophilic bone globules" on the same section.

Nothing is known as yet about agents which might influence the course of neurolathyrism; up to a year ago this was also true of osteolathyrism. It had been thought that the skeletal changes characteristic of the latter are due to an unchangeable chemical action of the lathyrogenic compounds upon some bone constituent, because it was impossible to influence osteolathyrism significantly by any dietary or pharmacologic measure. It is of utmost importance to establish whether a disease is amenable to therapy or to aggravation by "conditioning factors," despite the continued presence of the eliciting pathogen. If osteolathyrism were irresponsive to treatment, there would be little hope of finding any remedies against possible counterparts of osteolathyrism that we observe in man (degenerative arthritis, slipped epiphyses, Paget's disease of the bone, etc.). It would also be unlikely that any change in our external or internal environment (e.g., local or systemic stress, dietary factors, infections) could significantly participate in the pathogenesis of diseases related to osteolathyrism.

It was therefore of special interest to us when we noted that, in rats, the skeletal changes normally produced by feeding L. odoratus seeds or synthetic lathyrogenic compounds can be increased by STH, the somatotrophic hormone,

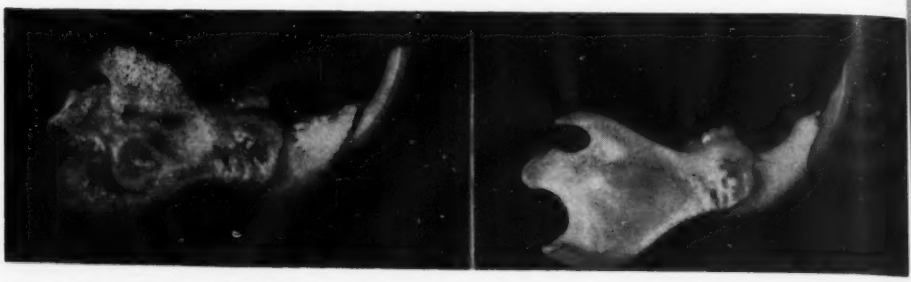

Fig. 4. *Adaptation to AAN.* Mandible of a rat treated with 150 mg/kg/day of AAN, per os, during 76 days. Note numerous exostoses, especially at muscle-insertion sites, and deformation of the tip of the incisor tooth (left). Compare with the virtually normal incisor of a rat treated with the same amount of AAN during the same period, but after two months of adaptation during which time gradually increasing amounts of AAN (10 mg/kg/day to 150 mg/kg/day) were given.

thyroidectomy, or partial hepatectomy, kernechtrot, alizarin, exposure to systemic stress, while they are suppressed by treatment with ACTH, cortisol, estradiol, thyroxin, hypophysectomy or mere inactivation of the bone owing to denervation.[18, 19] (Figs. 1 and 3.)

It has been noted that the syndrome of osteolathyrism can be qualitatively altered by desoxycorticosterone. This mineralocorticoid does not significantly affect the skeletal changes induced by lathyrogens but it predisposes to the formation of aortai aneurysms. By combined treatment with cortisol and desoxycorticosterone, it has even been possible to reverse the normal morphologic syndrome of experimental osteolathyrism; this corticoid combination caused lathyrogens to produce aortal aneurysms in the virtual absence of bone lesions, and under conditions in which bone lesions would normally have occurred in the absence of aortal aneurysms. Furthermore, it has been possible to induce a predisposition for giant callus formation at the site of bone fractures by pretreatment with lathyrogens in the rat.

Finally, following prolonged treatment with gradually ascending doses of AAN, it is possible to induce an extraordinary degree of resistance to this compound in the rat. The mechanism of this adaptation remains to be determined[20] (Fig. 4).

All these investigations have clearly demonstrated that both the intensity and the localization of the changes induced by lathyrogenic compounds are largely dependent upon conditioning factors. If metabolic defects, similar to those of lathyrism, occur in some common

Fig. 5. *Prevention by STH of vitamin-A overdosage.* Mandible of normal (top), vitamin-A-treated (middle) and vitamin-A + STH-treated rat. Note that hypervitaminosis-A resulted in several perforations within the flat part of the mandible and in absorption of the coronoid and condyloid processes with disappearance of the angle of the jaw. All these changes have been completely prevented by STH.

Fig. 6. *Sensitization of the skeleton to vitamin-A overdosage by cortisol.* Mandibles of rats treated with vitamin-A alone (left), cortisol alone (middle) and vitamin-A + cortisol. Here, the bone absorption is particularly pronounced in the rat that has been treated with cortisol and vitamin-A (the coronoid and condyloid processes as well as the angle of the jaw have virtually disappeared and the shaft of the bone is perforated in several places).

maladies of man, it should be possible to use the experimental disease as a model for the study of those factors which could affect the course of such clinical conditions. The hope that this might be the case supplied the stimulus for our investigations on lathyrism and for the compilation of a review, the main purpose of which is to show where further research appears to be most promising.[5]

Hypervitaminosis-A.—Since osteolathyrism is characterized by excessive bone proliferation, it seemed of interest to establish whether an experimental disease, in which bone absorption is the outstanding feature, could also be influenced by hormones. It is a well established fact that overdosage with vitamin A can produce extraordinarily intense bone absorption,[21] often accompanied by spontaneous fractures, multiple hemorrhages, and intense loss of weight.

It was found that, in the rat, cortisol and estradiol can greatly sensitize the skeleton to the characteristic manifestations of hypervitaminosis A, while somatotrophic hormone and methyltestosterone counteract the corresponding effects of an excess of vitamin A.[22-24] (Figs. 5, 6.)

Our findings show, furthermore, that in the syndrome of vitamin A intoxication—as in so many previously studied diseases—we have yet another example of a non-inflammatory morbid condition, in which a change in the hormonal milieu can abolish or accentuate susceptibility to a non-hormonal pathogen.

SUMMARY

Some of the organ changes produced by aminonitriles (skeletal lesions, nervous derangements, ocular changes) and by hypervitaminosis A (bone absorption, spontaneous fractures, multiple hemorrhages, and intense loss of weight) closely resemble certain spontaneous diseases of man. The discovery that the experimental lesions can be significantly influenced by external agents raises the hope that a better understanding of the factors which affect the experimental osteolathyrism and hypervitaminosis A may help to elucidate the pathogenesis, and to devise procedures for the treatment of the corresponding maladies of man.

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ACKNOWLEDGEMENTS

These investigations were performed with the aid of a grant from Geigy Pharmaceuticals and of Grant Laboratories. No. A-1641 (RI) from the National Institute of Arthritis and Metabolic Diseases, U. S. Public Health Service.

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RECENTE PROGRESSOS IN LE RECERCAS DE STRESS

Dr. med. Hans Selye

SUMMARIO IN INTERLINGUA

Depost le description in 1936 del syndrome de adaptation general (le responsa del corpore a stresses nonspecific), numerose investigationes esseva publicate in omne le mundo, tentante clarificar le mechanismo per le qual, in varie circumstantias, stresses pote producer o etiam prevenir morbos. In particular nostre Instituto se ha effortiate a provar que le hormones pote jocar un rolo decisive in le disveloppamento de plure morbos nonendocrin, i.e. de morbos non primarimente debite a disrangiamentos que ha lor origine in le glandulas endocrin mesme.

Certe alterationes producite in le organos per aminonitriles (lesiones skeletic, disrangiamentos nervose, alterationes ocular) e per hypervitaminosis A (absorption de osso, fracturas spontanee, hemorrhagias multiple, e perdita extreme de peso) resimila multo certe morbos spontanee del homine. Le discoperta que le lesiones experimental pote esser influentiate significativemente per agentes externe leva le sperantia que un melior comprehension del factores que affice le osteolathyrismo e le hypervitamininosis A experimental pote adjutar a elucidar le pathogenese e a perfectionar procedimentos pro le tractamento del correspondente morbos human.

SYNDROMES OF THE HEAD AND NECK OF DENTAL ORIGIN: I. PAIN CAUSED BY MANDIBULAR DYSFUNCTION*

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INTRODUCTION

The history of dental practice, during the waning years of the 19th century and the first two decades of this century, concerned itself primarily with advances in the mechanics of procedure. As a consequence, until a relatively short time ago dentistry focused most of its attention on the individual teeth and the structures in which they were invested. Recently, however, a welcomed change in point of view and practice has taken place. Dentists are broadening their outlooks and realizing that replacement of one or more missing teeth, by bridges or partial dentures, does not accomplish all that is desired in itself. This can no longer be regarded as the most complete service that dentistry can render the patient.

Today, we have come to the realization that the masticatory apparatus, which is comprised of the teeth, their investing structures, the temporomandibular joints, the neuromuscular mechanisms, and adjacent structures, is not an entity in itself but bears an important relation to the rest of the organism. The common meeting ground for all the specialties of dentistry is the study of occlusion. The utilization of the basic sciences such as anatomy, physiology, histology, and pathology in conjunction with clinical practice and experience are the basis for a clear understanding of the normal and the deviations therefrom.

The purpose of this paper will be to set forth the latest developments concerning derangements of the masticatory apparatus, the syndromes thereby produced, and their diagnosis and treatment.

The current literature contains many controversial interpretations of various aspects of this topic, and because the authors themselves have stressed the need for better correlated knowledge, it is no wonder that the general practitioner is more confused than ever. It seems that a clarification of joint structure, neuromuscular function, and their possible influence on auditory function is necessary in order to establish the possible relationship between mandibular malpositioning and various head and neck syndromes. Let us before discussing the subject define a syndrome as: "The aggregate of symptoms associated with any morbid process, and constituting together the picture of disease. The term is employed especially, though not exclusively, in relation to the grouped symptoms of disease of the central nervous system."[1]

THE PROBLEM

Costen[2-5] described his symptom-complex as occurring uniformly in disturbed temporomandibular joint function. The symptoms were described as follows: 1) *Ear symptoms,* such as loss of hearing, stuffy sensation in the ears, buzzing tinnitus, earache, and dizziness without nystagmus; 2) *Pain* in the form of headache about the vertex, occiput or behind the ears, and a burning sensation in the throat, tongue, and side of the nose; 3) *Miscellaneous symptoms* such as dryness of the mouth, herpes of the external ear canal and buccal mucosa; and 4) *Trismus.*

It was Costen's belief that all these symptoms were caused by altered temporomandibular joint function, due to overclosure of the jaws resulting from loss of posterior teeth. He maintained that pain arose as a result of pres-

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*This study was aided by the John Hartford Foundation of New York, and by the Murry and Leonie Guggenheim Foundation Institute for Dental Research, New York University College of Dentistry.

sure on or near the chorda tympani and the auriculotemporal nerves. Other authors[6-8] also reported improvement of symptoms following the restoration of a normal relationship of the condylar heads within the glenoid fossae by the technique of raising the bite. Although widely practiced at first and still in vogue today, Costen's form of treatment as well as the existence of the syndrome itself has been questioned.[9-11]

Unquestionably, no single factor can be responsible for all these varied symptoms. It is the *interaction* of the nervous, muscular, and vascular systems, at least in the affected region, which requires our attention. Further investigation should enable us to define more accurately the detailed symptomatology and its relationship to the various nervous and vascular networks.

Pain and dysfunction in the form of reduced mandibular movements were the symptoms found most frequently in several hundred cases by one investigator,[12] while the symptom-complex described by Costen was not found. The symptoms in these cases were precipitated by the sudden or continuous stretching of the masticatory musculature: yawning, excessive biting, prolonged sessions of dental treatment, or proprioceptive changes caused by sudden or extensive alterations in the dental occlusion through treatment. Female patients outnumbered the males with a ratio of 4 to 1. Treatment was primarily *spasmolytic,* consisting of ethyl chloride spray on the affected musculature and of intramuscular infiltration with a local anesthetic, followed by exercises of the masticatory muscles.

A recent study[13] conducted for eleven years on 899 selected cases of temporomandibular arthroses with pain showed that 551 patients benefited from dental care. In this study the ratio of females to males was 3.5 to 1. A distinctive though variable pain-pattern emerged from this study, as twenty different groupings of pain distribution were noted. This type of pain was concentrated at the origins and insertions of muscles, and the joint was found to be the commonest site. Treatment was primarily *prosthetic,* employing a temporary acrylic splint to discover the optimal occlusal level, followed by permanent occlusal reconstruction after close observation over a suitable period of time.

In both studies the pain described by the patients was a constant, *dull ache* as differentiated from the sudden, sharp, and intermittent pain of the neuralgias. Pains were also reported remote from the temporomandibular joints, namely in the jaws, neck, and shoulders.

THE NEED FOR FURTHER STUDIES

There appears to be a widespread belief that the problems of Costen's syndrome, or of temporomandibular dysfunction in general, are subject to controversy. Many otologists feel disinclined to deal with such problems. Yet, dentists frequently report good therapeutic results which cannot be brushed aside.

Partly to satisfy our own curiosity, and partly with the hope of helping to clarify this problematic matter, we decided to investigate it once again. We also had a good practical reason for a systematic study: many patients are seen in our clinics whose complaint of ear ache or other cranial pain cannot be satisfactorily solved with routine otolaryngologic management. At least some of these patients, it was felt, may represent cases of mandibular dysfunction.

In view of the complex variety of the symptoms commonly associated with Costen's syndrome, we decided to approach the problem from three distinct viewpoints, namely the complaints of 1) *pain,* 2) *ear noises, and* 3) *hearing loss.* Otherwise, the evaluation of symptoms, treatments, and results may become too involved to be convincing. For practical reasons, we decided to begin with a study of pain associated with certain abnormalities of the masticatory mechanism. The problems of tinnitus, hearing loss, and other associated symptoms will be the subject of subsequent investigations.

DIFFERENTIATION OF CRANIAL PAIN

When dealing with the emotionally tinged problem of pain, it is good to recall certain

basic facts of neuro-physiology. At first, we have to be sure of what we are talking about. Although all pain sensations are ultimately transmitted by nervous pathways, they may originate in various structures. Pain affecting the regions of head and neck can be caused by pathologic processes involving the nerves, the muscles, or the connective tissues. Among these, muscle pain appears to be of special importance for our subject. Therefore, we have to give some thought to the physiology of the masticatory musculature.

Whenever a patient suffering with pain around the face consults his family physician, he tries to determine the etiologic factors. If the cause is found, it is treated accordingly, and the symptoms subside. However, there are many cases which do not respond, and it is in these that the *dentofacial factors* are often overlooked.

Generally speaking, the factors causing pain about the head or predisposing to it are many. Nutritional or deficiency disease can impair the metabolism of the tissue cells, making them more susceptible to injury of a type that could give rise to pain. Other deficiencies such as in carbohydrates and oxygen also predispose to metabolic disturbance. Once a focus of local pathology has become established, it usually irritates the surrounding nerves, causing pain.

Some of the common causes of *nerve pain* are: direct trauma; indirect trauma on the perineural tissues causing pressure; traction pressure on the vascular supply, leading to anoxia and ischemia; extension of inflammation from neighboring tissues; invasion of a nerve by specific organisms or by a neoplasm.

When *muscles* are traumatized, compressed, overexercised, or in a state of sustained contraction or hypertonicity, they react with characteristic pain which is often accompanied by *spasm*. Muscles may be subject to pain as a result of deficiencies, such as in carbohydrates, or from the accumulation of products of metabolism as a result of muscle function.

Connective tissues, such as deep fasciae, tendons, ligaments and periosteum may be the location of pain resulting from excessive traction, or from injury from neighboring inflammatory processes. With regard to bone, the periosteum is very sensitive to traction tension. Cancellous bone is less sensitive to noxious stimuli, and compact bone hardly at all.

It is the task of careful examination to differentiate between these different types of pain. As a general rule, *neuralgic pain* is recognized by demonstrating tenderness at the points of exit of sensory nerves, such as at the trigeminal foramina. *Myalgic pain* is characterized by localized tenderness of the affected muscles and by their spastic contraction, such as in myalgia of the sternocleidomastoideus which hurts while being pinched. *Periosteal pain* shows sensitive trigger areas which may also appear thickened to the palpating finger.

GENERAL CONSIDERATIONS OF MUSCLE PHYSIOLOGY

The body contains various types of muscle fibers which fall into three distinct groups, namely: skeletal, smooth and cardiac. The muscles associated with functions in the head and neck region are composed entirely of skeletal muscle fibers.

When dealing with muscle pathology, one must bear in mind certain physiologic characteristics of skeletal muscle such as chemical action within the muscle fiber, its nutrition, and the phenomena of fatigue and tetanus. One of the most important facts is that skeletal muscle functions properly only when the normal distance is maintained between the origin and insertion of the fibers. Striated *muscle loses tone* and strength when the distance between these two points is shortened. On the other hand, *muscle tone is increased* beyond normal when the distance between origin and insertion is increased.[6]

Muscle has inherent properties, those residing in the muscle itself and others controlled by impulses reaching it from the nervous system. Both play an important role in considering the resolution of the problems of dental pathology. Any given muscle may on different occasions act (1) as a prime mover, (2) as an

antagonist aid in coordination, or (3) as an antagonist to neutralize an unwanted movement produced by another prime mover.

Muscles can contract either isotonically or isometrically. When they contract *isotonically,* they shorten but retain equal tension; if they contract *isometrically,* they increase tension but do not shorten and thereby retain their length. By contracting isotonically, muscles act as movers. By isometric contractions they act as holders, stabilizers, or positioners. In addition, muscles contract in preparing for their subsequent relaxation in order to brake or balance the moving contraction of their antagonists.[14]

ANATOMICAL REMARKS CONCERNING THE MUSCLES OF MASTICATION

The muscles of the face are arranged around each orifice in a perfect functional manner. This includes a sphincter and an opposing dilator arrangement which are specific for each orifice. In the eyelids and the lips these antagonistic systems are well developed, while around the nostrils and particularly around the ear lobes they are vestigial in man.

There is general agreement regarding the elevating action on the mandible by the temporal, masseter, and medial pterygoid muscles; about the retrusive action of the posterior fibers of the temporalis; and about the anterior-posterior and lateral movements of the mandible by the simultaneous contraction of the medial and lateral pterygoid muscles on one side.

Confusion can result from analyzing muscles outside their true functional groups, whereby the primary action of a muscle can be overlooked and some undesired secondary effect misinterpreted as the primary action. Further confusion arises from the fact that during forced movements muscles not normally in contraction may be brought in as accessory muscles. Any basic study should refer to normal tranquil movements if we are to understand abnormalities upon further examination. A review of the literature has revealed that no complete agreement has yet been reached on the following problem.

The suprahyoid and infrahyoid muscles require accurate and more comprehensive analysis if we are to understand their actions. Last[19] drew attention to the fact that muscles anatomically in contact do not necessarily share a common action: "In the functional sense, there is no such group as the 'suprahyoid muscles' and Brodie,[15, 16] McCollum,[17] Moyers,[18] and others fall into error in assigning a common action to muscles whose only common property is, in fact, that they lie together above the hyoid bone." Last further states that the suprahyoid group is not a group at all, but consists of muscles with very different actions. The digastric muscle helps to depress the mandible; the mylohyoid elevates the floor of the mouth; and the geniohyoid and stylohyoid determine the length of the floor of the mouth. They are all either opposed or assisted synergically by the infrahyoid muscles whose actions are of little importance to our present discussion.

Muscles are arranged in the body according to the best engineering principles, thus providing for conservation of energy. Furthermore, advantage is taken of effective leverage and assistance available from gravity. The following muscular functions influence the dental mechanism, namely; posture maintenance, deglutition, mastication, and facial expression.

With regard to the problem of *posture maintenance,* we may consider the shoulder girdle, clavicula, sternum, and scapula as the fixed base of operations. The head may be said to virtually teeter on the atlanto-occipital joint. Since the center of gravity of the head lies in front of the occipital condyles, it naturally follows that definite force must be applied to hold the head erect, a force which is provided by the large muscles of the back of the neck. Several groups of muscles are attached directly or indirectly to the anterior part of the head whose functions tend to add to the force of gravity, and thus to the load on the posterior cervical muscles, requiring thereby greater bulk and strength on their part. The most important of these anterior cranial muscles are the masticatory, the supra- and the infrahyoid groups. They constitute a sort of chain: with the man-

dible and hyoid bone, to which they are attached, they join the cranium to the shoulder girdle. It follows that within this chain the movements of the mandible and hyoid bone can occur independently of each other and of this functional chain itself.[16]

Last[19] agrees that the head is supported by the posterior cervical muscles, but he differs with Brodie on those muscles that are the opponents of the extensors. Brodie describes them as a chain that extends from the skull to the pectoral girdle and consists of the muscles of mastication that elevate the mandible, the suprahyoid muscles, and the infrahyoid muscles. Last believes that Brodie has completely ignored the prevertebral flexor muscles (rectus capitis, anterior and lateralis, and longus capitis) and the sternomastoid, which are the true flexor muscles of the skull on the spine.

"Brodie quotes Sherrington's Law of reciprocal inhibition of muscles but he misapplies it in his assessment. The tonus of his anterior chain of muscles is that of its weakest link. If the suprahyoid muscles invoke a contraction of the temporal and masseter as they do during the elevation of the floor of the mouth in swallowing, they likewise invoke a relaxation of the infrahyoid muscles. Thus, the head would be jerked back into extension each time we swallow if the infrahyoid muscles were indeed a factor in normal head posture."

Last[19] continues his discussion by stating that contraction of the temporal and masseter muscles certainly accompanies the mylohyoid stage of swallowing, but for a very different reason from that assumed by Brodie. The mylohyoid is organized in two halves which together form a gutter-shaped sling beneath the tongue. Contraction of the muscle elevates the tongue, making the floor of the mouth more shallow. This elevates the tongue, and if the teeth are held in occlusion, produces an increased pressure in the tongue, forcing it back into the oropharynx. This is the normal mechanism of the first stage of swallowing. Increased pressure on the tongue can be produced by mylohyoid contraction only if the mandible is held in occlusion by its elevators. For otherwise, the midline raphe of the mylohyoid would be fixed below the tongue and the contracting fibers of the mylohyoid would pull the mandible down.

STRUCTURE

Without correct structure there cannot be correct function. This, of necessity, leads to the formulation of three theorems:

1) Structure influences function — Wolf's law;
2) Structure is modified by function — Hooke's law;
3) Structure and function are affected by and affect the neuromuscular system.

If a study of the functional or mechanical adaptation of the skeleton were made, the bones of the face would be of great interest because of the marked difference between the upper and lower jaws and the presence of pneumatic cavities in the upper facial region.

Sicher[20] states that the *mandible* has a thick layer of compact bone, which is further strengthened along the lower border. The *maxilla,* which is anchored to the base of the skull, gains its reinforcement in certain areas from three strong pillars, the zygomatic, pterygoid, and canine. These are not straight because they must curve around the nasal cavity and the orbit, necessitating horizontal connections which act as buttresses. Of these three pillars the pterygoid process of the sphenoid bone is most important because its lower end is joined to the maxillary alveolar process by the interposed palatine bone; while its upper end is anchored directly to the base of the skull. Between the trajectories formed by the pillars, the bones remain thin and form the walls of the various cavities. The normal nasal septum is more or less straight, the turbinates are in their correct relationship and in correct position in the skull. The pneumatic sinus cavities in the upper facial region act as a partition and as weight reducing spaces between the supporting parts of the skull.

ETIOLOGY OF CRANIO-FACIAL PAIN

As every practitioner knows, the etiology of pain in the area of the head and neck com-

prises a great variety of causative factors. Instead of an endless enumeration, suffice it to say that the possible causes may be divided into three main groups: 1) *General systemic disease,* such as cardio-vascular, renal, or arthritic; 2) *Local pathology,* such as in the ears, nose, sinuses, throat, or cervical spine; and 3) *Abnormalities of the masticatory mechanism,* the topic of our present discussion.

Dentists are used to the following classification of oral conditions which may give rise to pain: Dental malocclusion with or without malposture of the jaw; Loss of teeth and the failure to replace them; Loss of vertical height between the jaws; Poorly fitting dental prosthesis; Faulty habits such as pipe clenching or unilateral mastication; and Disease of the temporomandibular joints. A frequent further cause of facial pain is the muscle imbalance due to malposition of the mandible, occlusal prematurities, and muscle spasm.

Several investigators have attributed the pain associated with temporo-mandibular joint disturbances mainly to the masticatory muscles.[21-23] In such conditions the pain and limited activity are associated with "trigger areas." These are small areas in the myofascial structures which are hypersensitive to stretch and give rise to muscle spasm with referred pain. It has been stated[24] that excessive opening of the mouth, as occurs in a yawn or during prolonged dental visits, sudden pain in the teeth, or severe pain in the area of the mouth, can evoke a compensating extended contraction similar to the action of a stretch reflex of other skeletal muscle. This would result in a prolonged tonic spasm of the jaw muscles such as is found in bruxism. Since there is no relaxation, the resulting dysfunction of the temporo-mandibular joint produces more pain, leading to a vicious cycle.

We must always bear in mind that malposition of the jaws does not necessarily result in pain or other disturbances. Whether or not such conditions occur depends not only on the presence of abnormal stresses or strains, but also on the adaptive capacity of the individual. This quality varies from one person to another, and it also varies with age, health, emotional status, etc. This accounts for malocclusions of considerable extent in some persons not causing any symptoms, while the slightest deviations in others result in extreme discomfort.

Wolff[25] has stated that when the pain sensitive structures about the head are stimulated, the muscles of the head contract. "If this stimulus be short-lived, muscle efforts are also brief. If, on the other hand, the painful stimulus from deep structures persists, long lasting contractions of the muscles of the head and neck and even of the jaws and face take place. Such sustained contractions result in pain and tenderness of the muscles involved."

SYMPTOMATOLOGY OF PAIN CAUSED BY MANDIBULAR DYSFUNCTION

In evaluating the causes of facial pain, we have to analyze the subjective complaints and the objective findings. The following *subjective complaints* are of importance. Varying degrees of discomfort from a dull sensation to extreme pain. The duration of this pain may be intermittent or quite constant. The pain may not be restricted to the site of the trouble but may travel other pathways.

Other *associated symptoms* may include: Subjective ear noises; Headaches of the migraine variety, affecting chiefly the occipital or vertex region; Clicking in the temporo-mandibular joint while chewing; Dizziness; Difficulty with swallowing; Tingling or burning sensations of the side of the tongue; and various other complaints which may not immediately appear to be related to the occlusal difficulty, such as impaired nasal respiration.

Upon examination one may detect any of the following *objective findings:* Crepitus in the temporo-mandibular joints; Stiffness, asymmetrical movements, or hypermotility of these joints; Locking of the jaws with ankylostoma; Muscular spasms; or impaired speech when the front teeth fail to occlude for the correct articulation of the sibilant sounds.

METHOD OF INVESTIGATION

At the New York Eye and Ear Infirmary, Ear, Nose and Throat Department, the resi-

dents have been taught to recognize the principal types of mandibular pathology, such as dental malocclusions, deviations of the depressed mandible from the midline, and displacement of the heads of the condyles in their fossae.

All patients visiting the clinic are first carefully examined for possible ear, nose or throat disease of local or systemic origin. Those patients presenting any of the symptoms discussed above and not suffering from any other illness are then referred for dental consultation.

Audiograms are taken whenever the patient complains of earache, ear noises, or hearing loss. The frequency and intensity of discrete tinnitus are measured in the customary manner. These permanent records permit objective comparisons of any therapeutic results.

Dental examination takes place right in the ENT clinic for the benefit of constant consultation between all interested specialists. This examination includes the search for tender muscle regions or "trigger areas." Simple treatments, such as ethyl chloride spray or procaine infiltrations, are given at the same time, if necessary.

DIAGNOSIS OF DENTAL AND MANDIBULAR PATHOLOGY

Dentistry now has reached that stage of development where it is ready to aid the otolaryngologist and other medical specialists in the differential diagnosis of head and neck syndromes. In addition, it offers scientifically sound corrective procedures for restoring the proper position of the jaws in the three dimensions of space.

It is obvious that a precise and complete diagnosis is essential before any therapeutic measures are undertaken. The dentist requires the definite exclusion of all local pathology not pertaining to his field. Again, a large variety of possible causes have to be considered by his referring advisors.

1) *Aural, nasal, and nasopharyngeal disease.* It is necessary to rule out the existence of more probable nasopharyngeal conditions, such as lymphoid hypertrophy, cysts, or tumors. Audiometric tests should be taken to determine the precise type of hearing loss that may exist.[26, 10] The dentist must also be advised what portion of a detected perceptive loss may be due to physiologic presbyacusis.

2) *Adenopathy,* especially of the parotid gland.

3) *Disorders of neural origin,* such as isolated or associated paralyses of cranial nerves.

4) *Collagenous disease,* like rheumatoid or osteoarthritis, and scleroderma which may effect the temporo-mandibular joint.

5) *Bone dyscrasias,* including osteoporosis.

6) *Traumatic disorders,* such as fractures and sprains of the mandible or its joints.

7) *Neoplasms* involving the temporo-mandibular joints; and

8) *Psychogenic factors.*

After this exclusion of other possible factors, a comprehensive dental examination must be undertaken to determine whether intradental, periapical, or periodontal lesions may exist. At the conclusion of his examination the dentist can present his opinion on the possibility of mandibular dysfunction. The following procedures will enable us to arrive at a final diagnosis:

1. Complete medical and dental *history*

2. *Symptomatology* (including pain): (a) Location (b) Quality (c) Onset and duration (d) Effect of environmental factors (e) Associated conditions

3. Full mouth *Radiographs:* (a) Intra-oral (b) Extra-oral — Cephalostatic temporomandibular pictures

4. *Study models* for three-dimensional analysis: By utilizing three anatomic planes of orientation, namely, the horizontal, mid-sagittal, and transverse, it is possible to establish reference points from which deviations from normal can be more readily observed.

5. *Inspection:* With the patient sitting upright in the dental chair without the headrest, or standing in a relaxed position, we notice any discrepancies in facial symmetry, head posture, indications of mouth discomfort, abnormal swallowing habits, speech difficulties, and deviations from the mid-line in opening and closing the jaws.

6. *Palpation:* Locate the areas which are painful and which of the muscles might be overdeveloped. Place the little fingers in the ear canals to determine whether there is any backward thrust of the mandible after the first contacts of the teeth are made, and if it causes the patient pain or discomfort.

7. *Auscultation:* Using a stethoscope to determine whether there is clicking or crepitus in the joints upon opening and closing.

TREATMENT

It appears that the most important problem of our deliberations is to determine which mandibular position, or range of positions, is best for our patients. We must utilize all diagnostic dental techniques, and proceed along certain definite lines.

First, we must correct the occlusion by eliminating premature contacts or other traumatogenic factors, if they are considered to be a possible cause of the mandibular dysfunction.

Next, the patient should be relieved of pain as soon as possible. Generally, the pain seen in these cases is due to muscle spasm and definite trigger areas can be palpated. These areas are sprayed with ethyl chloride[27]. We may also inject the muscle with normal saline solution, or with a local anesthetic solution[28]. A Luer lock syringe with a 21 gauge needle is used so that it is possible to aspirate before injecting.

Since the vast majority of the patients presenting painful head and neck symptoms (without otolaryngologic disorders) have malocclusions, temporary mandibular occlusal splints in conjunction with myofunctional therapy[29] have to be instituted. Not only will the vertical dimension between the jaws change, but the mandible will be capable of shifting to the right or to the left, of rotating to the right or to the left, of tilting in the horizontal and vertical planes, and of retruding or protruding in the sagittal line.

Whenever the occlusal dysharmonies have developed over a period of many years, it can be readily understood that certain degenerative changes may have occurred in the musculature. These range from fibrosis to contracture, and there are concomittant changes in habit patterns. In order to restore the patient to normal, new habit patterns have to be established. It is endeavored to return the patient to what we believe is their normal unstrained mandibular position. However, degenerative changes in the musculature and in the joints will of necessity alter our course. Time, therefore, is an essential factor in the solution of the problem.

After cessation of symptoms, and when the patient has been comfortable for several months without evidence of further periodontal breakdown, the permanent restoration of his masticatory function can be undertaken.

DISCUSSION OF FINDINGS

Tables 1 and 2 show the results of our observation of 100 cases during the past year. The most frequent areas of pain were the internal pterygoid muscles and the temporomandibular joints. However, pain was found in other areas remote from the jaw region. Patients were rendered symptom free or showed marked improvement by the administering of ethyl chloride spray or procaine injections to those "trigger areas" in the muscle where there was spasm. Frequently, by injecting the internal pterygoid muscles there would be relief of tenderness and pain in the other affected muscles. Favorable results were obtained in some cases simply by correcting the occlusion by means of equilibration or by temporary acrylic splints with adjunctive myofunctional therapy. Other cases required a combination of both procedures. 70% of the cases seen were of the painful variety. The ratio of females to males in this group was 3.5 to 1. This agrees with previous observations[12, 13].

Although only 7 cases of bruxism were reported, many of the other patients either clenched, ground, or gnashed their teeth. A majority of the patients in this study were found to be unduly emotionally tense or upset.

Twenty-five percent of the cases seen complained of subjective ear noises. Correction of the jaw relationships by means of temporary acrylic splints was the method of treatment. The results obtained were of a subjective nature only, and showed that one half of those per-

TABLE 1

SYMPTOMS	DISPOSITION					RESULTS			
	No. of Observations	Referred to own Dentist for Treatment	Mandibular Occlusal Plastic Splints	Equilibration of Occlusion	Ethyl Chloride Spray and/or Procaine Inject.	Not Known	No Improvement	Improvement	Symptom Free
A. *Painful*									
Trigeminal Neuralgia	1	1				1			
Occipital Pain	3	2	1			3			
Facial Pain	8	6		2		7	1		
Headaches	10	8	2	1		8			2
Muscle Spasms	68	27	8	3	43	25	1	28	14
Cervical Pain	2				1	1		1	
Temporomandibular Joint Pain	44	27	6	2	11	29	1	7	7
Ear Ache	13	9	2			11	1		1
Trismus	2					2			
B. *Non-Painful*									
Tinnitus	27	16	11			15	6	2	4
Vertigo	5	3	3			3		1	1
Clicking Joint	6	4	1			4		1	1
Crepitus in Joint	6	6				6			
Bruxism	8	7				7		1	

sons selected for treatment noticed some improvement or were symptom free. Future studies of this problem and of conductive hearing loss that may be related to jaw positioning[26, 30, 31] will be carried out with due scientific control.

We concur with the findings of Schwartz[12] in finding that Costen's Syndrome does not occur uniformly in temporomandibular joint dysfunction cases. The various symptoms of this syndrome can occur either singly or in groups in those cases which were seen at the clinic.

Whenever possible, patients who had their own dentists were referred to them for the construction of partial dentures, full dentures, rebasing of dentures, and resetting of teeth. This accounts for the large number of cases in the 'Not Known' category at this time. Let us now examine a few typical cases.

TABLE 2

THE FREQUENCY OF SPASM IN THE MUSCLES OF THE HEAD AND NECK

Internal Pterygoids	48
Masseters	30
Temporals	22
Sternomastoids	11
External Pterygoids	6
Posterior Cervicals	6
Digastrics	5

CASE REPORTS

1. A.P., 35 year old white female was seen at the Clinic in May 1958. She complained of pain of one year's duration in both temple areas; pain in both temporomandibular joints; pain in both sterno-mastoid muscles, (near their origin); pain in the right and left pterygoid muscles; and headaches every 2nd day in the frontal and temporal regions. Medical and otolaryngologic findings were negative.

Upon dental examination, occlusal disharmonies were evident due to malpositioned teeth. A temporary acrylic splint was inserted early in June 1958 to correct the malocclusion. The right and left temporalis muscles and both temporomandibular joints were sprayed with ethyl chloride and the other affected muscles were injected with xylocaine whenever necessary during the course of treatment. The patient was symptom free by the end of August 1958 and remained so to date.

2. J.M., 34 year old white male presented himself to the Clinic in May 1958. He complained of pain since November 1957 surrounding the left ear which radiated into the mandible and to the upper lip. Most of the left side of his face was painful. The patient had infraorbital and frontal headaches which radiated to the vertex and down the back of the neck. He also complained of tinnitus in the left ear. His hearing was found to be normal. Marked trismus was noted, most probably precipitated by excessive clenching and bruxing. Medical and otolaryngologic examination showed no organic pathology.

The dental diagnosis revealed a case of marked anterior overbite, collapsed posterior occlusion due to missing first molar teeth in the mandibular right and left quadrants, and resultant muscle spasms. The painful muscles were either sprayed with ethyl chloride or were injected with xylocaine. A splint was inserted on the 14th of May 1958, and within two weeks the patient was feeling much better. He was also instructed to exercise his jaw muscles by passively and actively opening his mouth. Several times during the next few months symptoms reappeared, but with much less intensity. It was not until the end of September 1958, when he also changed his job, that the patient felt completely fit.

3. P.L., A 41 year old white female was seen late in November 1958. She complained of tinnitus in the left ear and dizziness which she first noticed in May 1958, and which had lasted for one month. Symptoms reappeared again at the beginning of November 1958 and occurred every day. She had pain in the left temporomandibular joint, left internal pterygoid muscle, behind the posterior border of the ramus of the mandible on the left, and the posterior cervical muscles. Her hearing was found normal and no organic pathology was noted on examination.

Dental examination disclosed missing teeth in the mandibular right and left quadrants, and the patient was wearing an upper partial denture for 17 years. The lower teeth were missing for 15 years and the upper teeth were missing for a somewhat longer period. The collapsed bite was corrected by means of a temporary acrylic mandibular splint which was inserted December 17th, 1958. The left temporomandibular joint was sprayed with ethyl chloride. The patient was symptom free five days later. She was able to return to work and travel by subway without a companion, and without getting dizzy.

4. L.S., A 40 year old white female was seen at the clinic in November 1958. She had pain of one year's duration in both temporomandibular joints (the left one more so), both internal pterygoid muscles, both masseter muscles, both temporalis muscles, the left external pterygoid muscle, the left sternomastoid muscle, and the posterior cervicals on the left side. The pain also radiated supraorbitally from the temple regions, resembling a headache. She also complained of an ache in the eyes.

The patient was without all four first molar teeth in the maxilla and mandible which resulted in a collapsed bite. A temporary splint was inserted on December 15, 1958 to correct the malocclusion. The patient was symptom free two weeks later.

5. N.B., a 22 year old white female pre-

ented herself to the Clinic at the end of October 1958. Her chief complaint was a pulsating tinnitus in the left ear and a marked noticeable clicking in the left temporomandibular joint. Her hearing was found normal. She had an ovarian cyst operation a year before with intubation anesthesia. At time of examination she was in excellent health.

Dental examination revealed a mandible which was rotated to the left, and marked deviation of the mandible upon opening the mouth. A splint was inserted early in November 1958 to correct the rotation. The click disappeared, but the tinnitus is still present.

SUMMARY

During the past year observations were made at the New York Eye and Ear Infirmary of 100 cases of otolaryngological disorders of dental origin. The majority of these patients complained of pain in the head and neck region which were traced, for the most part, to certain "trigger areas" in the muscles of mastication. The most common sites for this pain were the temporomandibular joints and the internal pterygoid muscles.

Treatment of these cases was focused on physiological prosthetic management of maxillomandibular dysfunction. A high percentage of successfully treated cases was obtained with this method.

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SYNDROMES IN CAPITE E COLLO DE ORIGINE DENTAL

I. Dolores Causate per Dysfunction Mandibular

Dr. med. dent. Harold Gelb e Dr. med. Godfrey E. Arnold

SUMMARIO IN INTERLINGUA

Nos ha attingite le comprension que le apparato masticatori—consistente del dentes, del structuras que investi le dentes, del articulationes temporamandibular, del mechanismos neuromuscular, e del structuras adjacente—non es un entitate autonome sed se trova importantemente relationate con le resto del organismo. Le area in que omne le specialitates del dentisteria es communmente interessate es le studio del occlusion. Le utilisation del scientias fundamental—anatomia, physiologia, histologia, pathologia, etc.—in conjunction con le practica e le experientia clinic es le base de omne elucidation del norma e del deviationes ab le norma.

Le objectivo del presente articulo es presentar le plus recente disveloppamentos relative al apparato masticatori e su disrangiamentos, le syndromes producite per ille disrangiamentos, e lor diagnose e tractamento.

In le curso del passate anno, 100 casos de disordines otolaryngologic de origine dental esseva studiate al Infirmeria pro Disordines Ocular e Aural a New York. Le majoritate de iste patientes se plangeva de dolores in le region de capite e collo, traciabile—in general—a certe "areas precipitatori" in le musculos de mastication. Le sitos le plus commun de iste dolores esseva le articulationes temporomandibular e le interne musculos pterygoide.

Le tractamento de iste casos se concentrava super le attacco del dysfunction maxillomandibular per prostheses physiologic. Iste methodo de tractamento produceva un alte procentage de successos.

THE INFLAMMATORY DISEASES INVOLVING THE MUCOUS MEMBRANES OF THE MOUTH*

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The mouth can be a veritable treasure chest for the diagnostician. Dentistry embraces a multitude of conditions which are not familiar entities to the medical profession, and Dermatology adds to the sum total other dermatoses which also present problems of diagnosis. This discussion will consider but a few commoner entities routinely met with in Dermatology.

The mouth is an accessible orifice for inspection. It can be an open sesame to the astute clinician in making differential diagnoses of diseases of more profound origin. Today, unfortunately, we do not always follow the example of our astute medical forebearers who routinely examined the tongue and the mouth. The repeated dictum that the oral cavity mirrors the presence of and reflects many constitutional disorders should always be remembered. This, of course, does not deny that the mouth may have pathology in its own right. A visual appraisal alone poses great diagnostic hardship on the oral surgeon. We, in medicine, are permitted the privilege of a total examination of the patient, his skin surface and all of the body orifices, whereas the dentist must make a snap diagnosis by only looking into the mouth of the patient. This approach, imperfect though it may be, may give many valuable tips as to the patient's general health.

INFECTIONS

The interpretation of cultures taken from the mouth is at times difficult to many of us because, from the bacterial flora recovered, not all of the presenting organisms are necessarily pathogenic for the presenting conditions.

Bacterial, (Streptococcal and Staphlococcal)

Impetigo Contagiosa, with its stuck-on golden crust met with principally in young children but also as a pyogenic infection at any age, is a hazard to the practicing dentist. Contamination may occur to the operator and the dental work had best not be done in the presence of this troublesome condition. We have observed Impetigo Contagiosa encroching on the vermillion border of the lips but have not observed it within the buccal cavity.

An intriguing lesion from a similar pyogenic origin is called Granuloma Pyogenicum. This lesion may appear on any part of the skin or in any part of the mouth, including the tongue. Clinically, it resembles the Hemangiomas of childhood. Granuloma Pyogenicum represents the infected inflammatory response to trauma and, under the microscope appears like a vascular sponge. Its removal is simple but must always be complete. The central vessel supplying the bloody sponge-like tumour must be destroyed and whittling surgical procedures are ineffectual. Under a local anaesthetic the lesion is destroyed by a high frequency needle or by the cautery. The base is thoroughly curetted and the central vessel obliterated. Radium has also been used successfully but at greater expense as to time and also to the patient's pocket book. The simple procedure outlined above can be effected in five minutes if the operator does not mind working through a bloody field.

Viral Infections

In general, one can state that those who have worshipped too much at the shrines of Mars, Venus or Bacchus, or who have overexposed themselves in the presence of Sol are prone to invite the precipitation of viral infections. Certainly, the ever-present aphthous stomatitis may follow high living in all its forms. Herpes Simplex has the precipitating factor of photosensitivity and its contagiousness from one person to another can not easily be dismissed.

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* Presented before the 12th Annual Convention of the American Academy of Dental Medicine, Montreal, Que., May 29, 1958.

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Canker sores and Herpes Simplex Recurrens are attributable to allergic, endocrine, psychogenic, bacterial and, of course, to viral causation. In general, any factors contributing to a lowered body resistance predisposes to their occurrence. Is it any wonder that recommended cures for these above conditions are legion? Practically, we have found that the antiseptic mouth wash Clorapactin ws 60 is an excellent oral antiseptic agent for these and any other infected lesions of the mouth. Patients tolerate its unpleasant taste to obtain relief of their troubles from its use.

Aureomycin or Neomycin, combined with Hydrocortone in paints or water soluble vehicles as topical applications, are also very useful. We have found that dental Hydrocortisone ointment in Petrolatum base is difficult to use on the moist surfaces of the mouth. Hydrocortone throat lozenges, or the administration of the cortico-steroids, have controlled many cases of aphthous stomatitis and removed the painful aspects of the disorder. Administration of adequate doses of antihistamines sometimes gives additional comfort.

The old and tried treatment of multiple vaccinations with Smallpox vaccine should not be forgotten. Over a six or eight month period, vaccinations are re-administered irrespective of successful takes at monthly intervals.

Verrucae Vulgaris

The finding of the viral caused Verrucae Vulgaris is not uncommon in the mouth. The buccal mucous membrane, the gums, the tongue, as well as the palates, both hard and soft, have been seen to be sites of involvement. Warts in the mouth are not easy to diagnose because of their changed appearance due to their constant bathing with saliva. This moisture at times gives mouth warts the appearance of Condyloma Acuminata which are so commonly met with in other folds of the body. Warts in the mouth are a constant source of cancer phobia and, on their removal, a biopsy is advised to allay the patient's unfounded fears. Cure is best effected under a local anesthetic by means of excision followed by the curette and cautery.

Syphilis

Since the advent of Penicillin, Syphilis seems to be a vanishing entity, but surprises are still in store for the diagnostician who puts all his trust in "God's gift to the gay," that wonder drug, Penicillin. As Stokes oft repeated in the past, it is necessary to have a high index of suspicion and to consider as Syphilis any lesion which does not clearly fit into a clinical picture of a known common entity. Extra-genital chancres can occur on any surface in the mouth. Mucous patches which give the appearance of flat, slightly raised, or possibly ulcerated, whitish blotting paper-like lesions, are to be critically surveyed. Split flat hypertropic papules occurring at the angles of the mouth, with extension to the adjacent mucous membranes, invariably alerts one to the possibility of Syphilis. Strangely, Lues has a predisposition to appear on pre-existing plaques of Leukoplakia and may be manifested as a Verrucal type of over-growth. The finding of a perforation of the palate in our Northern Hemisphere inevitably means a Leutic infection and here, as pointed out by Stokes, the Wassermann reaction is frequently found to be negative. The T.P.I. Tests can be utilized when failure of darkfield and Wassermann have left the diagnostic problem in doubt.

Tuberculosis

Tuberculous lesions are prone to locate on the buccal mucous membrane and on the hard or soft palates, and generally represent an extension from a deeper-seated pulmonary laryngeal or gastrointestinal focus. These tubercular lesions are nodular, ulcerative or covered with a thin serous crust, and are indolent in character. The tip and sides of the tongue are the usual locations and these locations are of great diagnostic value in making a differential diagnosis. Today it is rare to find Tuberculosis Cutis extending into the mouth from the face.

Actinomycosis

Actinomycosis is still occasionally encountered and the actinomyces in our small series of cases have invariably gained entrance to the body through decayed teeth, with evidence later on the cutaneous surface. On rare occasions, it is

well to keep this diagnosis in mind, particularly if the face is involved with cicatricial tissue and displays sinuses discharging sulphur-colored granules in the pus.

Mycological Lesions

Thrush

The edentulous sucking will not be considered in this discussion. We are encountering more cases of moniliasis of the mouth, perhaps due to greater interest in mycology in our hospital. With the advent of the antibiotics, the resulting suppression of the normal bacterial flora by these agents has paved the way for an over-growth of moniliasis.

Moniliasis

The finding of Candida Albicans in the mouth should alert us to the possibility of a deeper involvement by the invader. Moniliasis does attack the lungs and gastro-intestinal tract. It is wise to have stool cultures done on patients showing extensive Moniliasis of the mouth, and to consider the possibility of a bloodstream involvement in the acutely ill patients. To dismiss the patient with a simple mouth wash is not good medicine. For local conditions, the use of a mouth wash of Aq. Zephiran, 1-4000, Sodium Propionate 1%, and the usual gentian violet preparations are helpful agents. Mycostatin washes, lozenges and tablets are the specific remedies presently available. It is wise to continue therapy for a period beyond the disappearance of the lesions in order to prevent relapses.

Perleche

This annoying condition follows the angles of the mouth and spreads in ever-widening circles from the lips to the cheeks. It was vigorously attacked in former days with strong anti-ringworm remedies. Cultures are likely to reveal the presence of monilia. The cure of Perleche is bi-part in most cases: first by the dentist in opening the bite to obliterate the skin folds at the angles of the mouth; and then by the physican by applying an ointment containing Hydrocortisone and Mycostatin. Vitamins play but little part in the eradication of Perleche.

Blastomycosis

Cases of North American Blastomycosis are not too uncommon in this northern region, but those under our observation have generally displayed signs of the Granuloma on the skin and we have not come across any occurring within the mouth. Invariably, after search, lesions have been demonstrated in the lungs or other viscera. Local therapy alone is not to be considered and our recent cases have received Amphotericin B by intravenous drip. Costello of Bellevue Hospital, N. Y., has indicated additional beneficial results from the injection of Amphotericin locally into the superficial lesions, as well as the administration of systemic therapy.

PAPULO-SQUAMOUS DISORDERS

Lichen Planus

Lichen Planus with its asymptomatic silvery spider-web, lace-like distribution may occur anywhere in the oral cavity but it has a predilection for the buccal mucous membrane. Its discovery by the practicioner should be kept a secret to himself. Knowledge of the presence of Lichen Planus in the mouth is a great source of cancer phobia to patients. It is well-known that the mouth lesions are always the last ones to clear away.

Arsenical preparations, anti-malarial tablets and the heavy metals are the main weapons we have to control the disease. Occasionally, the tranquillizers may help to soothe the unruly spirits, but, of course, focal infection and oral hygiene must be considered in the etiology of Lichen Planus. Chronic discoid Lupus Erythematosus on the lips may be confused with the silvery scaly lesions of Lichen Planus.

Chronic Discoid Lupus Erythematosus

The main point of differentiation between Lichen Planus and Lupus Erythematosus is that the scales of the latter display follicular plugs and these lesions, after healing, tend to leave atrophic scars. Chronic Discoid Lupus Erythematosus is a troublesome condition made worse on exposure to sunshine but it is now well controlled by the administration of the anti-malarials and by the topical application of Hydrocortisone ointment. The sub-acute and the acute

disseminated varieties of Lupus Erythematosis should be given over to the care of a physician. In these latter conditions, great care is necessary in order that the patient's life may be preserved.

Seborrhoic Dermatitis of the lips is frequently mis-diagnosed as a Cheilitis, possibly arising from contact factors. It is not usual to think of dandruff as occurring anywhere else than on the scalp, but mal-function of the sebaceous glands and their proximity to pilosebaceous apparatus may give rise to dandruff scale on any hairy part of the body. The naso-labial fold, the angles of the eyes and the angles of the ears, may become involved in this fissured scaly dermatitis. Seborrhoic Cheilitis generally responds to ointments containing sulphur, Vioform and Hydrocortone. Riboflavin deficiency is sometimes lurking behind the scenes.

Psoriasis

The silvery scale disorder, with its familiar background, can and does on occasions appear on the tongue and lips. With modern therapeutic knowledge its removal is still difficult.

THE VESICULO-BULLOUS LESIONS

The vesiculo-bullous lessions of the mouth are difficult diagnostic problems. The common cause of vesiculation of the mucous membranes, other than those caused by contact substances, noxious drugs, allergens, or of Herpes Simplex, is Erythema Multiforme.

Erythema Multiforme

The causative factors of Erythema Multiforme may be the toxins elaborated by bacterial, food, drug or viral origin, and represent an allergy of the skin surface, or of the mucous membranes, to a circulating toxin derived from these sources. The seasonal occurrence and photo-sensitivity are well-known additional precipitating factors. It is rare to have Erythema Multiforme in the mouth alone; confirmatory evidence can be seen on other areas of the body. When the mouth is involved in the process, the breath becomes foul and eating and swallowing may be difficult. Mild general malaise may accompany the attacks of Erythema Multiforme. It is, however, the severe form of Erythema Multiforme, called Stevens-Johnson syndrome, which gives great worry to both patient and doctor. In this condition, the eyes, nose, genitals and the skin take part in the severe toxic-allergic process. The cortico-steroids are a proven remedy for Erythema Multiforme. In certain cases of Stevens-Johnson syndrome this modality is employed. However, cases for steroid therapy of Stevens-Johnson syndrome must be selected with care and, unless the patient is covered with an umbrella of antibiotics to control the infective process, one may encounter complications.

Pemphigus

The cortico-steroids are almost specific for the treatment of Pemphigus and, in the last seven years, our group has had great success in treating some thirty cases of Phemphigus with the cortico-steroids. During our first experience with this wonder drug, we lost four cases with a fulminating type of Pemphigus due to inadequate initial dosage. The findings of flaccid bullae in the mouth, with eroded red painful bases, mean complete bacterial studies must be done to rule out Vincent's Angina and other types of Stomatitis, but it is well known that the primary lesions of Pemphigus do sometimes occur in the mouth with no evidence of involvement on the skin surface. In the old days, lesions of Pemphigus appearing in the mouth or on the eyes carried with their appearance a grave prognosis but, since the advent of the cortico-steroids this outlook has been greatly modified.

Dermatitis Venenata in the mouth, from primary or allergic agents, is a whole chapter apart from this discussion. These lesions exhibit in most cases an erythema or a vesicular response to the allergens and ulceration of the mucous membrane is not uncommon. Antibiotic preparations, aspirin and aspirin gums, dentifrices, all antiseptics, local anesthetic and a denture, with many other substances, make up a large proportion of the agents causing trouble in the oral cavity. One must have the mind of a detective to search out the clues to discover the offending culprit.

MISCELLANEOUS DISORDERS

Dermatitis Medicamentosa

It is but on rare occasions that one sees fixed eruptions from sleeping tablets or from laxative medicine occurring in the mouth. Bismuth Stomatitis and Bismuth Lines are seldom encountered since the advent of Penicillin. Dermatitis Medicamentosa occurring on the mucous membrane represents an allergic response to drugs inhaled, injected, ingested, instilled or absorbed by the percutaneous route. To run the offending drug to earth, it is necessary to take a careful history of all drugs taken by mouth and to question the patient clearly regarding all over-the-counter and patented medicines used. Occasionally, it is wise to review the various items used in our own therapy for fear that something given with all good intentions may have caused a side effect.

Fordyce Disease (Pseudo-Colloid of Lips)

This is a nevoid congenital abnormality displaying pin-head to white-yellowish elevations appearing on the cheeks, lips and gums, and even on the genitals. It is of cosmetic but of no other significance to the patient. After several attempts in the past to destroy individual lesions by cautery, I have refused to treat patients with this condition. If they occur in a woman, she is fortunate enough to be able to cover them with lipstick; in the case of a man, my advice to him is that he had best learn to live with this minor disability.

Glossitis Areata Exfoliativa

Here one is dealing with a tongue bald of papillae and the bald areas are in geographic outline not unlike the condition occurring on the scalp, called Alopecia Areata. Nerve strain is supposed to be the causative factor of Alopecia Areata, and I am wondering if Glossitis Areata Exfoliativa might not fall also into this category. Except for mouth washes and the possible giving of vitamin tablets in combination with iron, I do not feel that one is justified in giving X-Ray as we used to do in the past.

Lip Biters

Some nervous individuals have the unfortunate habit of biting their lips and the resultant eroded mucous membrane at times takes on the clinical appearance of a Leukoplakia. Most of the lip biters we have met freely admit their habit but are not too keen on abandoning the practice.

Glossydynia

Patients with burning tongues are certainly a bane to the Dermatologist. They all have cancer phobia and not a few of them have been relieved of their symptoms by injecting small doses of Hydrocortone injectable into the affected area. At times, one is tempted to consider the diagnosis of galvanism, but who, as a Dermatologist, is in the position to offer advice to the dentist regarding dissimilar fillings in the mouth. Invariably, it seems that patients complaining of these burning sensations have had excellent dental care, and display to you a mouth completely overhauled. I fear to get into the mining field and give advice that all fillings should be dug out to relieve the symptoms. Patients may have to part with more gold in an attempt to relieve the source of the trouble with the possibility of ending up in a worse state.

Mucous Retention Cysts

A useful tip in the handling of Mucous Retention Cysts is to aspirate the cyst content and then inject Hydrocortone Injectable into the sac. This procedure is not always successful, but in many cases the cysts have disappeared without the need of surgical intervention.

The study of medical disorders, as reflected in the oral cavity, can prove to be an interesting hobby to the dentist. A team of consultants can be grouped together around the oral surgeon. Such a team should be composed of: a bacteriologist, a mycologist, a histopathologist, physician, a surgeon and even a lowly dermatologist, and, by group consultation, they may better be able to unravel the mysteries of the complicated cases. The exact study of mouth lesions can be fascinating. Let dentistry and medicine pool their efforts in this common project!

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Subdepartment of Dermatology,
Royal Victoria Hospital,
Montreal, Quebec

MORBOS INFLAMMATORI AFFICENTE LE MEMBRANAS MUCOSE DEL BUCCA

Dr. med. Lemuel P. Ereaux (C.M.)

SUMMARIO IN INTERLINGUA

Le bucca pote esser un ver tresoreria pro le medico diagnostic. Le dentisteria imbracia un multitude de conditiones le quales non es entitates familiar al profession medical, e le dermatologia adde altere dermatoses al summa total, le quales etiam presenta problemas de diagnose. Iste discussion considera solmente un micre numero del entitates le plus commun que es incontrate routinarimente in le dermatologia.

Le bucca es un orificio accessibile al inspection. Illo pote esser un "Sesamo aperi te" al astute clinico in facer diagnoses differential de morbos de origine plus profunde. Hodie, infortunatemente, nos non semper seque le bon exemplo de nostre astute ancestres medical qui routinarimente examinava le lingua e le bucca. On debe semper rememorar se le dicto familiar que le cavitate oral reflecte multe disordines constitutional. Isto, naturalmente, non nega le facto que pathologia del bucca mesme pote etiam esser presente. Un evalutation solmente visual impone grande difficultates diagnostic super le chirurgo oral. Nos medicos gaude del privilegio de un examine total del patiente, le superficie de su pelle e omne le orificios del corpore, durante que le dentista es obligate a facer un diagnose immediate per le mer inspection del bucca del patiente. Iste necessitate, in despecto de omne le imperfectiones que illo comporta, pote dar multe preciose indicationes con respecto al stato general del salute del patiente.

Le studio de disordines medical, como illos se reflecte in le cavitate oral, pote devenir un avocation interessantissime pro le dentista. Un equipa de consulentes pote gruppar se circum le chirurgo oral. Un tal equipa pote includer un bacteriologo, un mycologo, un histopathologo, un medico, un chirurgo, e etiam un dermatologo. Per consultation intra le gruppo illes va esser melio preparate a disintricar le mysterios del casos complicate. Le studio exacte de lesiones oral pote esser fascinante. Que le dentisteria e le medicina junge lor effortios in iste projecto commun.

THE DENTIST AND THE ENDOCRINE PROBLEM SOME PHARMACOLOGICAL ASPECTS*

K. I. MELVILLE, M.D.†

It is now clear that local changes in the tissues of the mouth can result from various types of endocrinological disturbances, that is to say, from the effects of chemical substances which are released in the body either locally, or from some distant organ or tissues. However, there is little or no specific information regarding the chemical basis of most of these abnormal oral manifestations.

From the point of view of the effects of drugs, it has long been known that following prolonged administrations of various heavy metals—arsenic, bismuth, mercury, and lead—characteristic changes can develop in the gum and mouth. These however, appear chiefly due to local nutritional changes, and it is now well established that most of the actions of these metals in the body, are concerned with their affinity for SH groups, which are present in various tissue enzymes essential for cellular metabolism.

Regarding the effects of the metal ions on SH containing enzymes, as is well known these can be strikingly antagonized by the compound: dithiopropanol (Dimercaprol or B A L). This effect again is based upon the high affinity of these metal ions for SH groups, and dimercaprol itself contains two SH groupings, as follows: $CH_2.CH.CH_2OH$. Indeed, these groupings combine with the heavy metal ions thereby removing them from the tissue. The combination with the dithiol is a relatively non-toxic and soluble one and therefore readily excreted from the body in the urine.

It is also of interest to note that B A L destroys the action of insulin probably by reduction of the S - S group, and can protect rats completely against the diabetogenic and fatal effects of alloxan. B A L has also been shown to inhibit many different metal-containing enzymes (polyphenol oxidase, carbonic anhydrase, catalase and peroxidase) but it also inhibits some non-metal protein systems. It may therefore exert more than a single type of action.

Another more recently discovered antidote for heavy metals is the ethylenediamine tetraacetate (E D T A, Edathamil, Versene). This compound in the form of its calcium salts, exchanges its calcium ions for the heavy metal ions, a process referred to as 'chelation,' and the non-ionized water soluble 'chelates' which are formed are non-toxic and excreted by the kidneys. This might suggest that disturbances in ionic equilibrium in the tissues may also be important in the actions of the heavy metal ions.

It is also of importance to recall that many of these metal ions lead to capillary injury, and local vasodilatation. This tissue damage leads in turn to the release of histamine and other substances, but whether or not such secondarily released chemical agents are in any way responsible for the observed changes, is still unknown. The basic actions of these drug agents would therefore appear to be twofold (a) primary, i.e. effects on tissue nutrition or metabolism, and (2) secondary, i.e. effects resulting from mechanical injury or tissue damage leading to release of histamine and other substances. Other mechanisms concerned with allergic or hypersensitization reactions may of course also be involved.

It is obvious then that in the pharmacological approach to the problems of localized changes in the oral cavity, fundamental mechanisms of enzymatic, metabolic and nutritional processes operate, probably through some of the above-described types of mechanisms. However, further investigations along these lines would be necessary before any definite conclusions can be drawn.

Still another aspect of the problem concerns the local release of chemical agents associated

* Presented before the 12th Annual Convention of the American Academy of Dental Medicine, Montreal, Que., May 29, 1958.

† Professor and Chairman of the Department of Pharmacology, McGill University, Montreal, Que.

with the nervous control of some of the tissues of the mouth. As is known chemical substances such as acetylcholine, adrenaline and noradrenaline are released at the myoneural juncture of autonomically innervated tissues. While these are mainly concerned with smooth muscle structures and glands, it is conceivable that excessive release of these 'chemical transmitters' might be important factors in local tissue derangements. Obviously, drugs which block the effects of these substances might induce opposite actions. For example, (a) it is well-known that atropine and atropine-like drugs block salivary and mucous secretions, leading to dryness of the mucous membranes. This in conjunction with other factors, might be important in initiating local oral changes. Again (b) chlorpromazine the first of the tranquilizers to be introduced exerts anti-adrenaline and anti-noradrenaline action which might be important factors in regard to its influence. However, again there are no experimental studies on these questions.

Finally, within recent years another chemical substance known as 5-hydroxytryptamine or serotonin, has been shown to be present in various body tissues. Like histamine, serotonin has been reported to be released in antigen-antibody reactions and it has also been found (again in association with histamine) in the so-called 'mast-cells.' Indeed, changes in serotonin in brain tissue have been claimed by some workers to be responsible for the tranquilizing actions of the reserpine, but again it must be emphasized that the exact role of serotonin in the body is still obscure.

In regard to the oral changes associated with systemic drug administrations, a problem of special interest has been the occurrence of gingival hyperplasia, following the therapeutic use of diphenylhydantoin (dilantin) in the control of epileptic seizures. As is well-known some patients undergoing treatment with this agent develop gross disturbing overgrowths of the gum tissue. Histologically, this is associated with proliferation of capillaries and connective tissue and hyperplasia of the epithelial surface of the gums. It has also been observed that stopping the administration of the drug leads slowly to restitution to normal. The hyperplasia appears therefore to be of a benign or non-malignant type. There is no specific therapy for this condition. Good dental care appears to help but does not prevent it, and in cases of excessive hyperplasia, surgical excision by the dentist has often been resorted to. This, however, only affords temporary relief and with continued therapy remission ensues. During the past few years, my associate Dr. Lyman Francis and I, have been investigating certain pharmacological aspects of this problem, and I would like to indicate briefly some of our findings.

Despite the many theories which have been advanced during the past 20 years concerning the possible mechanism of production of gingival hyperplasia associated with diphenylhydantoin therapy in man, there has been surprisingly little experimental investigation of this problem. Indeed, in Drill's Pharmacology in Medicine, Second Edition, 1958, it is stated that "prolonged administration of anti-convulsant doses of the drug (diphenylhydantoin) in animals, fails to produce pathologic changes in the tissues." While this is still current opinion, it should be stated that in 1952, King reported that following prolonged administration of the drug to ferrets, both gross and microscopic changes resembling those occurring in man, could be observed. This worker however, suggested that these changes occurred only under special dietary conditions and could be accentuated by local irritation (feeding of bones in the diet).

In view of the above it appeared worthwhile to reinvestigate the effects of administration of diphenylhydantoin upon tissue changes in various laboratory animals, with special reference to those occurring in gingival tissue. Since the lungs, skin and mucous membrane are rich in histamine and serotonin, it was decided to undertake a comparative study of the "histamine and serotonin contents" of normal gingiva and of gingiva obtained from diphenylhydantoin-treated animals. While the exact roles of histamine and serotonin in tissues are still obscure, evidence exists that they play some part in anaphylactic and allergic responses, and although

it has been repeatedly suggested that the gingival changes following dilantin therapy in man, might be of an allergic nature, there have been no previous experimental studies in the literature concerning histamine and serotonin changes in the gingiva in this condition.

In regard to earlier experimental studies with dilantin, Merritt and Putnam (1938) reported no gross evidence of gingival changes in cats, dogs and rats following prolonged dilantin administration. Shafer (1948) also failed to detect either gross or microscopic gingival changes in rats despite administration of the drug for 13 to 14 weeks. In 1952, however, as already pointed out, King in England reported that following prolonged dilantin administration to ferrets both gross and microscopic changes resembling those previously seen in man, could be induced experimentally. Vichi and Masi (1955) have also reported some similar findings in rabbits, but these have not yet been confirmed.

Finally, in regard to the possible role of local irritation of the gums in this condition Swensen (1954) implanted stainless steel wires around the lower anterior teeth of rats, and after keeping the animals on dilantin for 3 months, could observe no significant influence of this type of local irritation upon the development of the condition. It would therefore appear that local irritation is not an important factor in the initiation of the hyperplasia, although it might obviously be responsible for superimposed secondary inflammatory changes.

In the first series of our experiments, no gross or microscopic changes could be detected in hooded rats, albino rats, hamsters or guinea pigs kept on the drug for periods as long as three months. Since in these experiments high doses were employed it was evident that these gingival changes were not simply direct acute toxic effects of the drug.

On the other hand, using both young and adult ferrets (22 animals) it was observed that all of the animals maintained on a normal diet with added dilantin showed progressively increasing gross evidence of varying degrees of swelling of the gingiva around the maxillary molars. With lower doses of the drug (40 mg. daily orally) changes began to appear about the 32nd week and culminated in visible overgrowth of the tissue as the experiment continued. With higher doses (200 mg. daily orally) the changes appeared somewhat earlier (about the 12th week), but were not essentially different and progressed rather like those from the smaller dose.

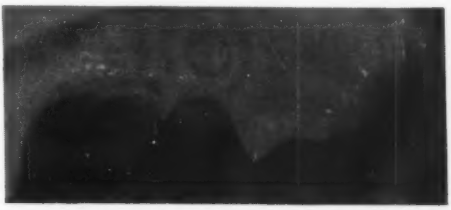

Fig. 1. Photograph of maxilla of normal untreated ferret, showing normal appearance of molar and premolar teeth, and gingiva.

Some typical examples of the gross and microscopic changes observed in ferrets after periods ranging from 20 to 60 weeks with oral doses of 40 mg. to 200 mg. per day are shown in the first few slides. Thus, Fig. 1 shows a photograph of the maxilla of a normal untreated adult ferret. It is to be noted that the dense sharp outlines of the molar and premolar teeth are clearly seen and the adjacent gums present a regular and uniform appearance.

In Fig. 2 is shown a similar photograph of the maxilla from a dilantin-fed adult ferret (40 mg/day) maintained on the drug for more than 1 year (60 weeks). As can be seen the adjacent gum tissue presents an irregular pattern, but there is a clear demarcation between

Fig. 2. Photograph of maxilla of dilantin-fed ferret, showing molar tooth partially covered and premolar tooth completely covered with hyperplastic gingival tissue. A dose of 40 mg. of dilantin sodium was administered orally daily for 60 weeks previously.

Thirteenth Annual Convention Committee: (Bottom Row, L. to R.) Drs. Harold A. Lentz, Reuben Feltman, Louis B. Kelsten, and Samuel A. Ament. (Top Row, L. to R.) Drs. James B. Dickinson, Frank H. Schambach, Mrs. Philip C. Lowenstein, Mrs. Harold R. Gelhaar, Mrs. Samuel Turkenkopf, and Dr. Harold R. Gelhaar.

Retiring President, Dr. [illegible]resents the [illegible] incoming Presid[illegible] G. Stewart

AMERICAN ACADEM[illegible] DE[illegible]

13th Annual Meeting • [illegible]laridge, [illegible]
May 14, 15[illegible] 17, 19[illegible]

Dr. Don C. Lyons presents a charter to the Missouri Section. Dr. Gunter Schmidt, President, accepts on behalf of the newly created section.

Dr. Don C. Lyons is the recipient of the Distinguished Service Award of the Academy which is presented by incoming President Dr. George G. Stewart.

Mrs. Phillip C. Lowenstein (holding white bottle) demonstrated and had audience participation in a hobby she has developed. Under her supervision Academy ladies transformed wooden match boxes into beautiful jewel boxes and knicknack containers.

Dr. Don C. Lyons presents [illegible]rtificate [illegible] Nathan Wachtel for the e[illegible]nt manne[illegible] year's Mid-Winter M[illegible]g was c[illegible]

nt, Dr. [illegible]resents the gavel to
[illegible] Preside[illegible] G. Stewart.

Academy ladies accompanied by several youngsters prepare to leave on a conducted bus tour which included visits to the Lenox plant, Renault and Son Winery and a surprise luncheon.

EM[illegible] DENTAL MEDICINE

• [illegible]laridge, Atlantic City, N. J.
14, 15[illegible] 17, 1959

Dr. Frank H. Schambach (left) and Dr. Reuben Feltman (right) Co-Chairmen of the 13th Annual Meeting, receive certificates of Merit from President Dr. Don C. Lyons.

Dr. Lawrence Staples, Annual Awards Chairman for many years, is the recipient of the Certificate of Merit presented by Dr. Don C. Lyons.

[illegible]presents [illegible]rtificate of Merit to Dr.
[illegible]r the e[illegible]nt manner in which last
[illegible]Winter M[illegible]g was c[illegible]nducted.

The new 1959-60 National Officers include: (L. to R.) Dr. Abraham Goldstein, Vice President; Dr. Irving Yudkoff, Editor; Dr. George G. Stewart, President; Dr. Walter I. Levine, Secretary; Dr. William M. Greenhut, Treasurer. Dr. Hermann Becks, President-elect, is not in this photograph.

Fig. 3. Photograph of maxilla of dilantin-fed ferret, showing partial obliteration of the crown of the molar tooth with lobulations of hypoplastic tissue between the molar and the premolar, and partial obliteration of the premolar tooth.

this area and the normal healthy tissue. There is also a partial obliteration of the molar tooth and almost complete obliteration of the premolar—only the dense cusps of the teeth can be seen. Finally, around both the molar and premolar, the overgrowths of the gums show definite lobulations.

Fig. 3 demonstrates a still more advanced stage of these gross gingival changes. This is a photograph from the maxilla of another dilantin-fed ferret, maintained on a high dose level (200 mg. a day) but for a shorter period (5 months). Here again, it is seen that the overgrowth of the gingiva adjacent to the teeth presents an irregular bulbous appearance and both molar and premolar are partly obliterated.

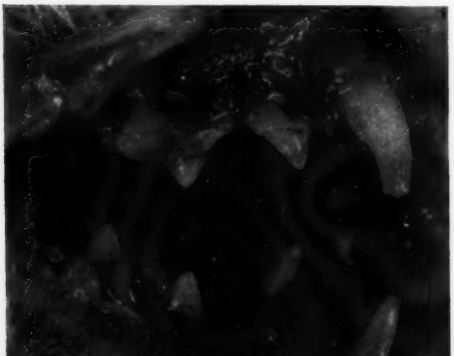

Fig. 4. Photograph (grossly enlarged) of jaws of ferret, showing crowding and poor alignment of teeth with hyperplastic gingiva. Dilantin started at age of 8 weeks, and continued for 60 weeks.

The areas between the teeth also show strikingly excessive downgrowths of the gingival tissue.

In regard to the gross changes observed in the gums of young ferrets (8 weeks old), it was observed that the changes were rather similar, although they started somewhat earlier than in the adults (about 12 weeks).

Fig. 4 shows a grossly enlarged photograph of both the upper and lower jaws of one of these animals. As can be seen, there is a crowding of the molar and premolar, and the teeth are in essentially poor alignment. The teeth are surrounded by hyperplastic tissue, with downgrowths of gingiva distal to the upper molar, and between the cuspid and premolar teeth. A similar picture was observed in all of the treated animals. On the other hand, as with the normal adult ferrets, the untreated controls in this series of experiments all showed regular evolution of dentition with normal appearance of the gums. These observations would seem to suggest that the fibrous state of gingival tissue might really be responsible for the observed experimentally induced malocclusion. It would therefore appear that malocclusion is a result rather than a cause of gingival hyperplasia under these conditions, and this interesting aspect of the work will be followed up in due course.

In Figs. 5 and 6, are shown some typical microphotographs obtained from sections of

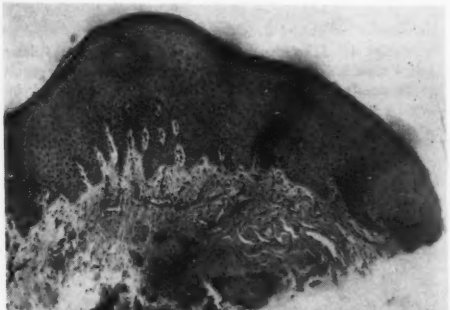

Fig. 5. Microphotograph of section of normal untreated ferret gingiva (same animal illustrated in Fig. 3), showing normal appearance of epithelium and deeper tissues. Magnification 90 X.

Fig. 6. Microphotograph of section of hyperplastic gingival tissue of dilantin-fed ferret (same animal illustrated in Fig. 5). Magnification 45 X.

gingiva in the above animals. Thus, Fig. 5 shows a microphotograph (90X) of a gingival section made from a normal untreated control ferret. As can be seen, the normal stratified squamous epithelium shows characteristic downgrowths, with fairly regular demarcation between the epithelium and underlying connective tissue. Special attention is drawn to the relatively few cells scattered throughout the subepithelial tissue layer.

In contrast Fig. 6 is a microphotograph (45X magnification) of gingival tissue obtained from a dilantin-fed ferret. It can be seen that there is a complete invasion of the underlying connective tissue by epithelium masses, which appear as isolated islands in several locations. The superficial layer of epithelium is also obviously relatively thickened and in some instances this was as much as 6 or 8 times that of the normal.

In regard to the microscopic examinations of other tissues, the skin, liver, spleen, and kidney showed normal histological appearances. On the other hand, there was some apparent increase in both the number and size of the lining epithelial cells in the bronchi and bronchioles. In these instances, the bronchial epithelial cells were markedly vacuolated, but their nuclei appeared normal and did not suggest an increase in mitosis. There is little doubt therefore that the gingiva possesses a curious almost specific sensitivity to these changes.

It therefore appeared that ferrets would be a satisfactory species for the study and it was therefore of interest to examine possible changes in histamine in the normal and hyperplastic gingival tisue of the ferret.

In this connection an extract was prepared from pooled hyperplastic gingival tissue of 4 ferrets and compared for its histamine-content with a similarly-prepared extract from 4 normal untreated ferrets. A total quantity of approximately 100 mg. of tissue was used for each extract. The procedure employed for extraction and assay of histamine in these and all other similar experiments was that described by Barsoum and Gaddum (1935).

The assays for histamine were carried out on the excised guinea pig ileum (approximately a one inch strip) suspended in a 4 ml. bath containing atropinized Tyrode solution. The bath was kept at a temperature of between 37° and 39° and aerated continually throughout the experiments. Histamine dihydrocnloride (Hoffman- La Roche) in aqueous solution was used as the standard. At the end of most experiments the effects of additions of the antihistamine agent mepyramine (0.1 ml. of 1.2 x 10^{-6} concentration), upon the responses to both the standard and unknown extract, were determined.

Fig. 7 shows the results of an assay of gingival extracts obtained from normal untreated and dilantin-treated ferrets, which had previously developed gingival hyperplasia. The re-

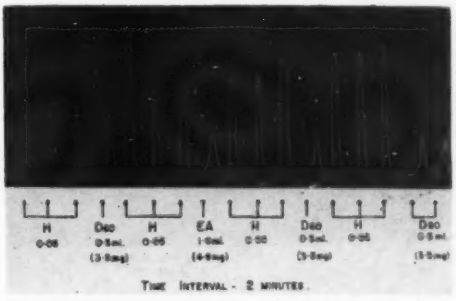

Fig. 7. Histamine assay on guinea pig ileum, showing effects of dilantin-fed ferret extracts (D60), and responses to 0.05 microgram of histamine (H/0.05), also, responses to extracts from normal control ferrets (EA). In these and all succeeding figures on histamine assays, an interval of 2 minutes elapsed between each test of extract or histamine.

sponsiveness of the gut was first standardized, using a 0.05 microgram dose of histamine (H/0.05), the injection was repeated 3 times and led to approximately the same responses. The gingival extract from the hyperplastic tissue of the 60-weeks dilantin-fed ferrets (D 60) in a dose of 0.5 ml. containing the equivalent of 3.5 mg. of tissue, led to a somewhat similar contraction, as that to the preceding histamine (0.05 micrograms) and appeared to be a typical histamine response. On the basis of this observation it was calculated that the gingival tissue of the dilantin-fed ferret contained approximately 14 micrograms per gram of histamine or of histamine-like substance.

It is also shown in the figure that following the initial test of the extract, the next subsequent test response to the standard histamine (H/0.05) was greatly potentiated. After two further standard tests with the histamine, responses similar to those previously observed were however obtained. At this point an injection of the extract prepared from the normal untreated animals was tested, using 1.0 ml. (EA) of extract, this contained the equivalent of 4.8 mg. of tissue. On the basis of this response the normal gingival tissue of the ferret was estimated to contain approximately 5 mu./gm. of histamine or histamine-like substance. When the standard histamine dose (H/0.05) was again tested there was also no definite potentiation, although succeeding responses were somewhat gradually increased. The record also shows that two later repetitions (end of tracing) of tests of the extract from dilantin-fed animals (D 60), induced quantitatively reproducible responses.

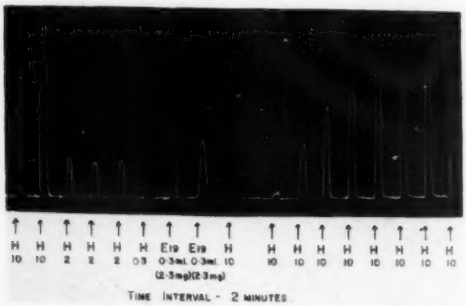

Fig. 8. Histamine assay on guinea pig ileum, showing effects of normal untreated dog gingival extract (E19), and response to 10 and 2 micrograms of histamine (H10 and H2) before, and 10 micrograms of histamine (H10) after, testing the extract.

It would appear from the above experiments, (a) that gingival hyperplastic tissue contains relatively higher concentrations of histamine or some histamine-like substance, than normal gingival tissue, and (b) that this change was associated with the appearance of some histamine-potentiating substance in the extracts of the hyperplastic tissue. However, the prolonged period of administration required to induce gross changes in the gingiva of ferrets, and the relatively small quantity of material that could be obtained from each animal, led us to extend our observations to other species of animals, and dogs were used in the subsequent experiments.

Fig. 8 shows an example of an assay of an extract from the gingiva of a normal untreated dog. Here again the normal responses to 10 microgram (H/10) and 2 microgram (H/2) doses of histamine are first shown. The preparation gave no response to a histamine dose of 0.3 micrograms (H/0.3). As can be seen two injections of doses of 0.3 ml. of normal gingival tissue extracts (E/19), induced a greater contraction than that obtained from 2 micrograms of histamine (H/2), but less than that obtained from 10 micrograms (H/10). The second response to the extract was however somewhat less than the first.

Furthermore, as in the case of the extracts prepared from normal ferrets, following exposure of the ileum to the extracts, the figure also shows that immediately thereafter, there was no response at all to a 10 microgram dose of histamine (H/10). However, with repeated washings and repetitions of histamine there was progressive restoration of the normal responsiveness of the gut (end of tracing).

In Fig. 9 is shown an example of another similar assay from an extract obtained from normal gingival tissue. Following four successive repeated tests of the extracts (E/13), there was a progressive diminution of the response.

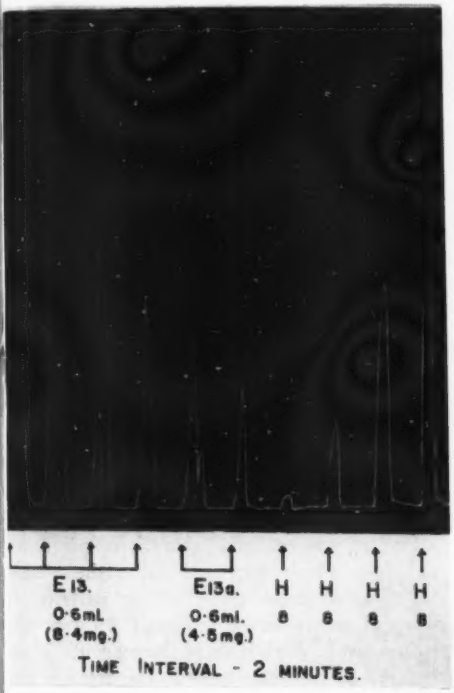

Fig. 9. Histamine assay on guinea pig ileum, showing diminishing effects of repeated tests of normal dog gingival extracts (E13 and E13a), also responses to repeated 8 micrograms test doses of histamine (H/8).

In addition, the record also shows two successive responses from an extract (E 13a) prepared from the gingivae taken from the opposite side of the mouth of the same animal. These again showed apparently progressive decreased responses on repetition. As in the previous experiment, again the preparation was almost completely non-responsive to histamine but on repetition there was progressive increased responsiveness, as illustrated in the previous figure. Similar results to those shown above have been observed in the large majority of experiments.

From the assay data obtained from 12 normal dogs, it was calculated that the quantities of histamine or histamine-like substance present in the gingiva ranged from 3 to 89 micrograms per gram of gingival tissue, with a mean of 22.4 micrograms per gram.

Extracts obtained from dogs kept on dilantin for short periods (up to 12 days) showed no significant difference from those of the normal untreated animals, however, after periods of 13 or more days of drug administration, similar changes to those previously described with the hyperplastic tissue of ferrets were consistently noted. Thus Fig 10 shows an assay in which after the initial standardized responses to a 1 microgram dose of histamine (H/1), repeated three times, a gingival extract from a 22 days dilantin-fed dog (D/22), in a dose of 0.6 ml. led to the striking response shown. Again on repetition, of the test with even a much smaller dose (0.2 ml.) of the same extract there was a considerably greater response than with the standard histamine doses of 1 microgram, (H/1).

It can also be seen that following these tests with this extract the responses to the standard histamine (H/1) are greatly potentiated. This

Fig. 10. Histamine assay on guinea pig ileum, showing effect of dilantin-fed dog gingival extract (D22), and responses to 1 microgram doses of histamine (H/1) before and after testing the extract. A dose of 200 mg. of dilantin sodium was administered orally daily for 22 days previouslv.

is in marked contrast to the diminished effects to histamine observed after tests of extracts made from normal gingival tissue. Indeed, even at the end of the experiment the histamine responses were still increased, and considerable time was generally required before return of the gut to its normal sensitivity to histamine in such experiments.

Fig. 11 shows the responses to an extract from dog gingiva obtained after 46 days of dilantin. As can be seen, the responses obtained from 4 initial successive tests of the extract (D/46) were quantitatively repeatable. A later addition to the bath of the same quantity of extract (D/46), towards end of record also showed an augmented response. In this experiment, the histamine responses recorded throughout are all identical and obviously maximal, so that no evidence of potentiation is demonstrable. It is, however, clear that unlike extracts from normal gingiva, the successive histamine responses are again definitely not diminished.

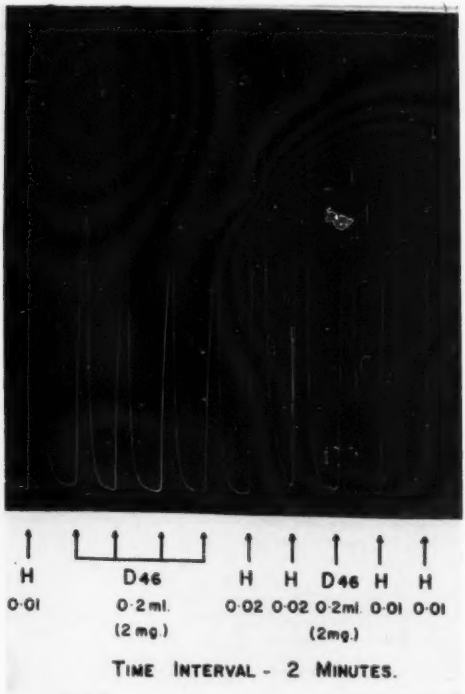

Fig. 11. Histamine assay on guinea pig ileum, showing repeated effects of a gingival extract from a dilantin-fed dog (D 46) and responses to 0.01 and 0.02 microgram doses of histamine (H/0.01 and H/0.02).

Finally, from the assay data obtained in tests performed on extracts from 12 dilantin-treated dogs, it was calculated that the gingiva contained quantities of histamine-like substances varying from 6 to 240 micrograms per gram of tissue, with a mean of 87.7 micrograms per gram.

Similar results to those shown above have been observed in most experiments. However, varying quantitative degrees in the changes described, were noted in different animals, although on dilantin for the same periods of time. It did not appear therefore, that these changes were related to or dependent upon either the dosage and the duration of treatment.

Similar studies have also been performed and dogs which were fed either the closely-related agent, mesantoin (100 mg. daily for 32 days) or phenobarbital sodium (100 mg. daily for 36 days). With both of these agents, it was observed that the responses to the extracts were identical to those observed with extracts prepared from normal gingival tissue, and it is therefore clear that these gingival tissue changes induced by dilantin were rather specifically related to dilantin administration. This was also confirmed in other studies in which no significant histamine changes were detectable in other tissues (lung, skin, kidney) of the dilantin-fed dogs.

In a third series of experiments, changes in the serotonin content of the dog's gingiva were similarly compared in normal and dilantin-treated animals. The extraction procedure employed was that described by Gaddum (1953), and the extracts were assayed by two different methods of serotonin assay, using the rat colon and the rat uterus previously sensitized by injections of stilboestrol as described by Dagliesh (1953).

Employing the rat colon method of assay, Fig. 12 shows the effects observed in comparing extracts obtained from the gingiva of a dog

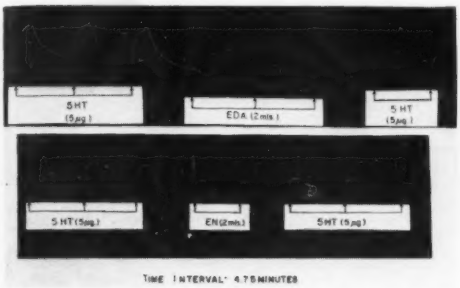

Fig. 12. *Above*: Serotonin assay on albino rat colon, showing responses to 5 microgram doses of standard serotonin (5-HT/5-μG), and the absence of responses to 2 ml. quantities of extracts from a dilantin-fed dog (EDA). *Below*: Serotonin assay on albino rat colon, showing responses to 5 microgram doses of serotonin (5-HT/5-μG) or normal untreated dog, (EN) and the standard (5-HT).

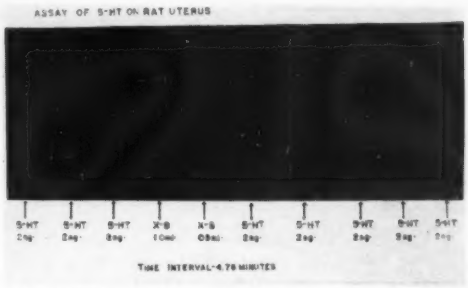

Fig. 13. Serotonin assay on the albino rat uterus, showing absence of responses to two successive tests of extracts obtained from gingiva of a dilantin-fed dog (X-B) and responses to 2 nonogram doses of standard serotonin (5HT/2 ng) before and after.

which had been maintained on a daily oral dose of 200 mg. of dilantin for 41 days, (EDA (2 mls.) upper tracing) with a gingival extract from a normal dog (EN (2 mls.) lower tracing). As can be seen following three initial 5 microgram administrations of the standard serotonin (5 HT/5 μg) almost equal contractions were recorded in both assays. Whereupon, little or no response was observed (upper tracing) following three successive additions of 2 ml. of the extract from the dilantin-fed animal (EDA 2 mls.), while two successive additions (lower tracing) of 2 ml. of extract (EN, 2 mls.) from normal gingiva showed definite responses. Subsequently, good responses to the standard serotonin solutions were also elicited in both experiments.

When the rat uterus method assay was used similar results were also obtained. Thus, Fig. 13 shows an assay using an extract obtained from a dog maintained on a dose of 200 mg. of dilantin for 34 days before the gingiva was excised. Again, it can be seen that following each of three initial administrations of 2 nanograms (0.002 mg.) of serotonin, there was an almost identical good contraction recorded (5 HT/2 ng.) in each instance. However, following two successive tests using 1 ml. and 0.05 ml. of the extract (X B) from the dilantin-fed dog, no responses were elicited. In view of the high sensitivity of the preparation to serotonin, it must be concluded that following dilantin there is a complete disappearance of serotonin from the gingiva.

The record however, shows that following the tests with the extracts there was a temporary diminished responsiveness of the uterus to serotonin, and the tissue was only slowly restored to normal sensitivity. This effect may be due to some type of serotonin-antagonism induced by the extract and possibly related to the complete absence of any response to the extract itself. This hypothetical material would however be extractable by the same procedure employed for serotonin. This aspect of the problem again requires further study.

Fig. 14 also shows results of a typical experiment on the rat uterus, and in which the responses to an extract prepared from the gingiva of a normal untreated dog were tested. Again, it is seen that three initial injections of a dose of 10 ng. (0.010 mg.) of a standard serotonin solution led to rather similar responses. However, following the addition of two successive doses of 0.2 ml. and 0.05 ml. of the extract (X/0.2 ml. and X/0.05 ml.) the contractions of the uterus were so intense that in both instances the record extended above the upper limit of the recording drum. Again by this procedure, it is clear that normal dog gingiva contains considerable quantities of serotonin.

In order to quantitate the effects of the extract smaller doses were then tested, and as can be seen a dose of 0.01 ml. of the extract (X/

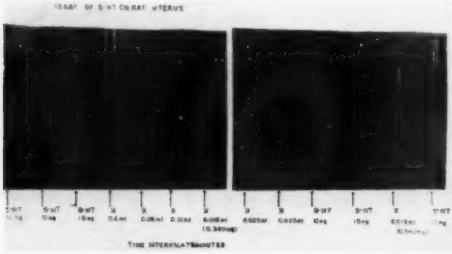

Fig. 14. Serotonin assay on albino rat uterus, showing responses to repeated tests of normal untreated dogs gingival extracts (X) and to repeated 10 nonogram doses of standard serotonin (5-HT/10 ng.)

0.01 ml.) gave a response which can be differentiated from that to a dose of 0.015 ml. of the extract (X/0.015 ml.). After a further interval of some minutes, uniform responses were then elicited by doses of 0.025 ml. of the extract (X/0.025 ml.). Following these additions to the bath, the uterus showed much greater responses to the standard serotonin, and the effects of two successive 10 nonogram doses (5-HT/10 ng.) were clearly greater than were observed at the beginning of the experiment. However, the response to the extract (X/0.015 ml.) was also somewhat increased but the exact significance of this apparent potentiation of serotonin action by the normal gingival extract is not clear.

From the above findings, there is a little doubt that normal dog gingiva contains a high concentration of extractable serotonin, and that following dilantin administration, this extractable gingival serotonin disappears completely. This latter effect also appears to be associated with development of some temporary antagonism to subsequent responses to serotonin.

Finally, some preliminary observations have also been made using three samples of gingival tissue, that were surgically excised from patients, through the kindness of Dr. Harvey. These were removed under local anesthesia (procaine) from patients showing excessive gingival inflammatory changes, but not receiving dilantin. After excision the tissue was immediately refrigerated and extracted as in the earlier studies. Fig. 15 shows that following initial standardization of the gut response to histamine (H/

Fig. 15. Histamine assay on the guinea pig ileum, showing the responses to human gingiva inflamed tissue extracts (HG/0.2 and H/0.4 ml.) and comparative responses to pig lung extracts (L/0.5 and L/0.2) and to standard histamine (H/0.1 and 0.2). At the end of the record, the absence of the usual responses to the extract (H/0.2) and to histamine (H/0.2) after mepyramine (AH) are shown.

0.1), addition of 0.2 ml. of the extract (HG/0.2 ml.) prepared from excised human gingival tissue (200 mg. extracted in 25 ml. volume) gave a positive contraction which was only about one half of that obtained from 0.1 microgram of standard histamine. This response moreover, was quite repeatable quantitavely, as shown at subsequent points marked (HG/0.2 and HG/0.4). It is also quite clear that extracts obtained from the tissue did not antagonize the effects of the histamine (H/0.1). Finally, as can be seen at the end of the experiment both the responses to the extract (HG/0.2) and that to histamine (H/0.2) are blocked by previous treatment with mepyramine (AH), indicating clearly that the effect of the extract was due to histamine.

In order to assess the relative histamine content of the gingiva in comparison with the guinea pig lung (which is known to be rich in histamine), and extract prepared from guinea pig lung tissue was also tested as shown at (L). As can be seen, after addition of 0.5 ml. of lung extract (L/0.5) prepared from 200 mg.

of tissue extracted in a volume of 25 ml., the contraction recorded was somewhat higher than that from 0.2 ml. of the gingival extract. However, the latter response was almost equal to that observed after addition of 0.2 ml. of the lung extract as shown (L/0.2). From these findings it may be concluded that the histamine content of inflamed human gingival tissue is at least twice that of the normal guinea pig lung tissue.

The above observations lend strong support to the concept that gingival changes following dilantin are due to some allergic-type of phenomenon, which leads to changes in histamine and 5-hydroxytryptamine (serotonin) in the tissue, but the exact role of each is unknown. These changes are also presumably associated histologically with increased mast-cell formation, which has been recently demonstrated by Dewar (1957). The question therefore arises as to whether or not similar changes in histamine and serotonin occur in other body tissues and if not, why is gingival tissue particularly susceptible to these changes?

In regard to the possible roles played by the amines, nothing definite can be said. It is however of interest to note that Cambel and Sgouris (1952) have reported that after repeated subcutaneous injections of histamine dihydrochloride (0.04 mg. daily) in rats, there was "a progressive thickening of the gastric mucosa due to an increase in size and number of parietal cells." Thus, after 4 weeks of histamine "enlarged parietal cells often replaced peptic cells which were much decreased in number, and were also compressed by them." The authors conclude that histamine can induce experimental hyperplasia of the gastric mucosa. However, as the authors pointed out there were many associated local and systemic changes, e.g. cell hypertrophy, decrease in peptic cells, decrease in size of spleen and some atrophy of the pancreas. The direct casual relationship of histamine to this type of tissue hyperplasia cannot therefore be stated.

Histamine is also well-known to be released into tissues following irritation and inflammation, and it is difficult to assess the role of such factors in this type of experiment. However, it has recently been reported by Gaillard (1957) from preliminary observations on 2 cases of dilantin hyperplasia, that administration of the anti-histamine agent chlorprophenylpyridamine (Teldrin) led to some favorable effects upon the hyperplastic overgrowth. Martin (1957) however, observed that while this agent appeared to lessen the associated superimposed gingivitis, it did not influence the tissue overgrowth. The possible usefulness of this and other known types of anti-histamine and anti-allergic drugs upon experimental gingival hyperplasia remains to be investigated and assessed.

In regard to the possible significance of the gingival changes in 5-hydoxytryptamine after dilantin, it is of some interest to note that in clinical cases of dilantin hyperplasia, pain is frequently absent. High histamine and serotonin concentrations have been reported to be associated with the local vasodilator and irritant effects (itchiness and pain) induced by certain materials like nettle sting (Emmelin and Feldberg 1947), wasp venoms (Jaques and Schacter 1954), etc. Armstrong et al (1953) have also suggested that while the "itchiness might be due to the histamine liberation, the pain might well be due to the 5-hydroxytryptamine." The clinical absence of pain in this condition might therefore be due to the above-described disappearance of the serotonin, but obviously this again requires further study.

It is also of some interest to note that studies concerning the influence of dilantin upon brain serotonin levels (Bonnycastle et al 1956) have shown contrarily that in rats treated with dilantin (100 mg/kgm twice daily, intraperitonely), for two days there is an increased concentration to twice the normal brain level. During this short period of treatment no gingival changes would of course be expected, but these observations tend to confirm some possible relationship between serotonin-changes and the effects of dilantin on the body tissue.

Finally, since little or no hyperplastic changes or alterations in histamine-content appear to occur in most of the other body tissues ex-

amined (except lung) it is possible that some local physiological condition in the gum and perhaps to a lesser extent in the lung, might be responsible for the apparent selective involvement of these organs. Moore (1958) has stated that "discrete pulmonary lesions which may be hypertrophy of the small pulmonary vessels and/or the bronchioles," have been observed at autopsy on individuals previously treated with dilantin. However, there has been no systematic investigation of this aspect of the problem. It also is not clear why there are apparent species differences in the gingival responses to dilantin. Both the ferret and the dog, however, showed similar changes in histamine so that the species differences are probably more apparent than real. This problem again needs more detailed study.

In conclusion, it is postulated as a working hypothesis that under the influence of dilantin the observed histamine and serotonin changes in gum, resulting from some allergic mechanism, might conceivably "trigger" cell processes concerned with mitosis. It is quite clear that the histamine and serotonin changes do not arise directly from the presence of dilantin in the body, but requires some time (14 days in dogs) before the initiation of these changes. Further investigations of the fundamental biochemical changes associated with hyperplasia may indeed throw further light upon some of the pharmacological problems concerned with the development and treatment of some of the oral changes referred to earlier, and perhaps also upon the obscure processes concerned with local inflammatory reactions and cell multiplication in general. Some of these problems are being further studied.

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ACKNOWLEDGEMENTS

This investigation was carried out in conjunction with Dr. Lyman Francis, while working towards his M.Sc. degree in Pharmacology, and with the technical assistance of Mrs. Barbara Koss Levine. A preliminary report of some of the findings has been published in the Journal of the Canadian Dental Association, V. 24, March 1958, pp. 142-147. This investigation was also supported in part by grants to the author by the Associate Committee on Dental Research, National Research Council of Canada.

LE DENTISTA E LE PROBLEMA ENDOCRIN: CERTE ASPECTOS PHARMACOLOGIC

Dr. med. K. I. Mellville

SUMMARIO IN INTERLINGUA

Il es nunc clar que le alterationes local in le histos del bucca pote resultar ab varie typos de disrangiamento endocrinologic, i.e., ab le effectos de substantias chimic que es liberate in le corpore, localmente o ab nu organo o histo distante. Nonobstante, il existe pauc o nulle information specific con respecto al base chimic del majoritate de iste anormal manifestationes oral.

Ab le puncto de vista del effectos de drogas, on ha longemente sapite que post le administration prolongate de varie metallos pesante—arsenico, bismuth, mercurio, e plumbo—alterationes pote disveloppar se in le gingivas e in le bucca. Nonobstante, istos es apparentemente debite principalmente a local alterationes nutritional, e il es nunc ben establite que le majoritate del actiones de iste metallos in le corpore es concernite con lor affinitate pro gruppos de SH, le quales es presente in le varie enzymas histic que es essential pro le metabolismo cellular.

Il es obvie, dunque, in le interpretation pharmacologic del problemas del alterationes localisate in le cavitate oral que un mechanismo fundamental de enzymatic processos metabolic e nutritional es active, probabilemente via le unes e le alteres del varie typos de mechanismo supra-describite. Nonobstante, investigationes additional de iste genere essera necessari ante que ulle conclusion definite pote esser derivate.

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JACK P. FACUNDUS College of Medical Evangelists, School of Medicine, 1720 Brooklyn Av., Los Angeles 33, Calif.	W. E. MacPherson, Dean, School of Medicine, College of Medical Evangelists.
ARTHUR GOLDSTEIN State Univ. of N. Y. Upstate Med. Cr., 766 Irving St., Syracuse 10, N. Y.	Davis B. Johnson, Dean, State Univ. of N. Y., Upstate Medical Cr.
*WILLIAM P. GRAHAM III Colorado General Hospital, Denver 20, Colorado	John McK. Mitchell, Assoc. Dean, Univ. of Pennsylvania School of Medicine.
GEORGE J. KORKOS 1623 N. 60 St., Milwaukee, Wis.	John S. Mirschboeck, Dean, Marquette Univ. School of Medicine.
*DAVID LAWRENCE 2907 Dumesnil St., Louisville 11, Ky.	J. Murray Kinsman, Dean, Univ. of Louisville School of Medicine.
BOBBY RAY RICHARDSON 1620 East Lamar St., Sherman, Texas	Robert W. Lackey, Asst. Dean, Univ. of Texas Southwestern Med. School.
KENNETH LLOYD MELMON 150 Kirkham St., San Francisco, Calif.	J. V. deC. M. Saunders, Dean, Univ. of Calif. School of Medicine.
LARRY D. SAMUELS New Windsor, Ill.	Granville A. Bennett, Dean, Univ. of Illinois College of Medicine.
*ROBERT IGNATIUS SCILEPPI 30 Warwick Rd., Rockville Cr., N. Y.	James O. Pinkston, Assoc. Dean, State Univ. of N. Y., Downtown Medical Center, College of Medicine.
DAVID A. SEARS 3720 S. W. Dosch Rd., Portland, Oregon	D. W. E. Baird, Dean, Univ. of Oregon Medical School.
JAMES KERMIT SEXTON 1307 Genevieve St., Pikeville, Kentucky	Manson Meads, Academic Dean, Bowman Gray School of Medicine.
DON B. SINGER 620 South 6th, Chicasha, Oklahoma	Stanley W. Olson, Dean, Baylor Univ. College of Medicine.
RONALD STEFANI 2642 N. Nordica, Chicago, Ill.	John F. Sheehan, Dean, Stritch School of Medicine at Loyola Univ.
DAVID SCHICK TRUMP 15808 Oakhill Rd., E. Cleveland, Ohio	Charles A. Doan, Dean, Ohio State Univ. College of Medicine.

* Recipient To Receive The Journal Only.

MEDICAL SCHOOLS — UNITED STATES (Cont'd)

Recipient	*Dean of School*
THOMAS TORGERSON 858 Woodland, Birmingham, Mich.	Gordon H. Scott, Dean, Wayne State Univ. College of Medicine.
ROGER G. VIETH 318 South Lincoln St., Westmont, Ill.	W. C. Davison, Dean, Duke Univ. Medical Center.
MARVEN H. WALLEN 217 E. Pine Ave., Wildwood, N. J.	H. J. Taggart, Dean, Hahnemann Medical College.
HANS WILHELMSEN 2817 Montebello Terrace, Baltimore 14, Maryland	Wm. S. Stone, Dean, Univ. of Maryland School of Medicine.
MISS JAYNE WUNSCH 4015 W. Alabama, Houston, Texas	John B. Truslow, Dean, Univ. of Texas Medical Branch.
RICHARD BUNG DICK CHUM 3362 Campbell Ave., Honolulu, Hawaii	Wm. N. Parkinson, Dean, Temple Univ. School of Medicine.

MEDICAL SCHOOLS — CANADA

JOHN STUART SOELDNER 16 Farnum Rd., Waltham, Mass.	C. B. Stewart, Dean, Medical School, Dalhousie Univ.
NORMAN FRETZ c/o Oran M. Fretz, R.R. #4, Stouffville, Ontario	G. E. Hoffs, Asst. Dean, Univ. of Western Ontario Medical School.
W. D. S. THOMAS 1787 West 49th Ave., Vancouver, B.C.	H. Rocke Robertson, Acting Dean, Faculty of Medicine, Univ. of British Colombia.
WM. JOHN FARLEY 141 Dundas St. West, Trenton, Ontario	G. H. Ettinger, Dean, Faculty of Medicine, Queen's Univ.
MRS. ANITA JAKEROW 131 Blackburn Ave., Ottawa, Ontario	J. Jacques Lussier, Dean, Faculty of Medicine, Univ. of Ottawa.
*IHOR DYRDA 7225 St. Michel Blvd., Ville St. Michel, Montreal, Que.	Wilbron Bonin, Dean, Faculty of Medicine, Univ. of Montreal.

MEDICAL SCHOOLS — ABROAD

RICHARD JOHN KIMBER, B.D.S. 3 Mariner St., Linden Park, South Australia	K. Stuart Wetzel, Dean, Medical School Univ. of Adelaide, North Terrace, Adelaide, S. Australia.
FRANCISCO MATA BENITEZ Prolongacion Lox Manolos, Qta. Kiva—La Florida, Venezuela	Carlos Gil Yepez, Dean, Escuela de Medicina, Ciudad Universitaria, Caracas, Venezuela.
RAJA NABIB KHURI American Univ. of Beirut, Beirut, Lebanon	Joseph J. McDonald, Dean, Medical School, American Univ. of Beirut, Lebanon.

* Recipient To Receive The Journal Only.

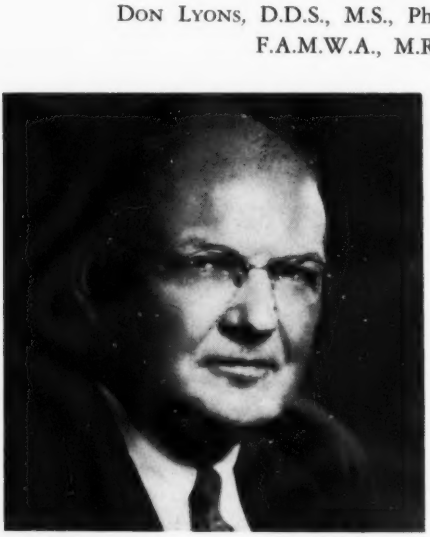
MEDICAL SCHOOLS — ABROAD (Cont'd)

Recipient	*Dean of School*
EVELYN G. ONG 786 San Sebastian, Quiapo, Manila, Philippines	Agerico B. M. Sison, Dean, College of Medicine, Univ. of Philippines, Manila, Philippines.
MISS ACELA GUTIERREZ Floridablanca, Pampaga	Lauro H. Panganiban, Dean, F.E.U. Institute of Medicine, Manila, Philippines.
BRIAN C. O'RIORDAN Turretts House, Charleville, Co. Cork, Ireland	J. C. Saunders, Dean, Faculty of Medicine, University College, Cork, Ireland.
R. C. DAWSON 20, Kent Road, Harrogate, England	W. Rae, Dean, Royal College of Surgeons in Ireland, Dublin, Ireland.
KATHLEEN LAGAN 157 Cregagh Rd., Belfast, Northern Ireland	P. J. Stoy, Dean, Faculty of Medicine, Queens Univ., Belfast, N. Ireland.
RUDOLF DORIZZI Seftligenstr. 289, Wabern-Bern., Switzerland.	Prof. Dr. A. Ott, Dean, Zahnarztliches institut dr Unverstitat, Berne, Freiburgstr. 7, Berne, Switzerland.

* Recipient To Receive The Journal Only.

RETIRING PRESIDENT'S MESSAGE

DON LYONS, D.D.S., M.S., Ph.D., F.A.A.A.S., F.A.P.H.A.
F.A.M.W.A., M.R.S.H. (Eng.)



It seems that every speaker I have listened to recently starts his lecture with a quotation. This is apparently a return to the old method of attention getting which was taught in public speaking classes some years ago, which is all right with me for I never could remember a good story long enough to get across its punch line. So I have selected a quotation which I think illustrates the motives of the members of the American Academy of Dental Medicine in their continual search for knowledge. The late Newton D. Baker was often a graduation ceremony speaker and is said to have started his talks by reminding his audience that, "The man who graduates today, who stops learning tomorrow, is uneducated the day after." That precept is even more pertinent today in our efforts to keep abreast of the tremendous flow of new knowledge that continually overwhelms us. The goals of this Academy have always been to make available to its members the opportunity to learn the newest and best methods of caring for their patients as is attested by the excellence of both our National and Section programs throughout the years.

This past year has been one of continued growth in many directions. We welcomed a number of new members in December and more at this meeting. We are extending a welcome to our newest Section, Missouri, at this time and expect others this coming year. We visited many Sections during the past year and found groups of enthusiastic members and the finest of clinical programs devoted to the concepts of dental medicine.

This year we were able to start a program of interchange of guest delegates with the Institute of Dental Medicine (the name has been changed to Oral Biology as of July, 1st.), thus achieving one of the fondest wishes of our Vice President, Hermann Becks. Last fall two of us were guests at their meeting at Palm Springs, California. We have two of their members with us today. I hope they have enjoyed our meeting as much as we did theirs and that we have been as good hosts as they were. We expect this mutual interchange to continue to the benefit of both groups.

The activities of the Committee on the Samuel Charles Miller Memorial have progressed to the point where they plan an annual memorial lecture and they have developed a commemorative medal to be given to the lecturer. The first lecture is to be given soon.

Your committee for the International Interchange of Dental Medicine has been working hard all year and has developed an international program in cooperation with the State Department. Plans are well under way to send a team of five members of the

Academy to Peru and to other countries for a period of two or more months at a time. All the basic work has been completed and only final approval and timing remains. Our Vice President is lecturing at the University of Bonn in Germany at the present time under a similar arrangement.

The activities of the Specialty Board have been held in abeyance this past year pending acceptance by the Council of Education of the American Dental Association. Following the meeting of the A.D.A. in Dallas another meeting was set up at the A.D.A. headquarters in Chicago last February. At that time representatives of the Boards of Endodontia, Pathology, Periodontia, and our group met. Plans were discussed regarding an area board and definite recommendations adopted for presentation at the Council next September. I have just received notice that another meeting is to be scheduled within the next few weeks. It is hoped that something more concrete can be presented at the December meeting.

Our Journal continues to climb to greater scientific heights and international recognition. It is the leader in the field of dental literature in its publication of abstracts in interlingua, one reason for its extensive foreign subscription list. We are planning a special issue for the Centenary of the A.D.A. in September, the issue will be available to all registrants of the meeting who desire a copy. Our advertising committee reports several conferences with ethical drug firms and feel very hopeful of obtaining some support from this source.

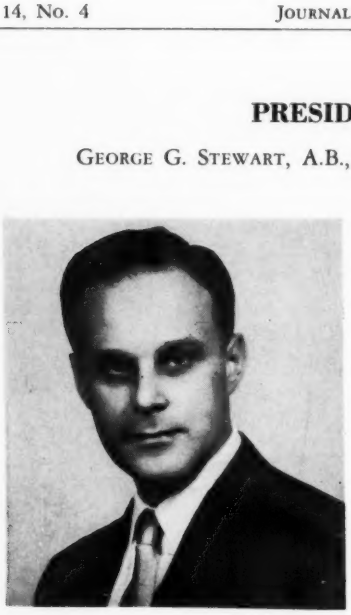
Our past midwinter meeting was exceptional not only in programing, but it broke all previous attendance records and was a decided financial success. The Committee is to be especially congratulated for its efforts on behalf the Academy. The 1960 annual meeting is scheduled for Philadephia. All initial plans have been completed for an outstanding meeting. We know from past experience that we can expect the best. Your National Convention Chairman is planning instituting a new venture in 1961. Planning is under way to make the 1961 Annual meeting a combined vacation and educational event with a boat trip to Bermuda. The scientific sessions will be held on the boat and there will be plenty of time to enjoy the warm breezes and soft sands of the Bermuda beaches.

The officers, committees, and the membership at large have been very cooperative and helpful this year. We have made gains, but let me quote another proverb, an old Latin one this time, "No gain is so certain as that which proceeds from the economical use of what you already have."

I have made many new friends throughout the country this year. It has been a real privilage and pleasure to serve as your President. Now let me turn the gavel over to our new President, George Stewart, who can be depended upon to carry the Academy on to greater things and consolidate the gains we have made this year.

PRESIDENT'S ADDRESS

GEORGE G. STEWART, A.B., D.D.S., F.A.C.D., F.A.D.M., F.A.A.A.S.



I would like to take this opportunity to thank the officers and members of the Academy for the honor they have bestowed upon me, by electing me President. I am mindful of the responsibilities, and will endeavor to execute the duties of the office, to the best of my ability.

I know that my term will be a pleasant one, because the men you have elected and those who volunteer to serve are capable and conscientious: and working together as a team, we will further the objectives of the Academy, and of Dentistry.

If we take the time to reflect for a moment on the history of organized Dentistry in this Country, we will note that the stature of our profession has been growing, because we have more, and better services, to render the public. Originally, emphasis in the practice of dentistry was on mechanics, and only at the turn of this century was biology introduced into our profession. Now the biologic aspects are being expanded to include the total organism, in his total environment. This is the essence of Oral Medicine, and Dental Medicine.

It is true that our prime concern is with the oral cavity, but the oral cavity is a part of the biologic unit—man. In this second century of organized American Dentistry, the American Academy of Dental Medicine must be the leader in furthering this concept, if we are to continue as a growing, vigorous profession.

We can help realize our objectives by encouraging the development of new sections, and keeping our present sections active. I believe that the parent organization should assume greater responsibility in providing speakers and clinicians to our sections, and that programing requirements be so altered as to permit the individual sections to function more freely. This might be fostered by establishing a "Sections Activity and Speakers Committee".

We might also extend the functions of the "Membership Retention Committee", by having this committee maintain contact with the recipients of the Graduate Awards. Since these individuals do receive the Journal for 5 years, why not also give them free junior membership for 5 years, as part of the award. In this way, they can automatically be invited to active membership, at the end of the 5 year period. During this "junior membership" they should be encouraged to participate in the activities of the section nearest their home.

We should further press our objectives, by enlarging our Journal, both in content and circulation. With the section on Interlingua we are reaching areas of the world where English alone would have little value.

I feel that we should continue to make every effort to send individual members, or

teams of our members, to less economically fortunate areas of the world, to help spread current teaching in the fields of Oral Medicine.

The human relationships thus established, might also foster better international understanding.

I think, too, that we should continue to support our American Board of Oral Medicine, in gaining official recognition by the American Dental Association, since the Board represents the means by which higher standards can be set for the teaching and practice of Dentistry.

Before closing, I would like to take this opportunity to invite the members and guests of the Academy to our next annual meeting, which will be held over the Memorial Day weekend in Philadelphia, from May 27th to May 30th, 1960. We plan to have an excellent program, from both the academic and social standpoints, and we hope to see you all there.

Academy Proceedings

AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES MID-ANNUAL MEETING

The American Academy of Dental Medicine will sponsor its fourteenth Mid-Annual Meeting and Luncheon at the Hotel Sheraton-McAlpin, on Sunday, December 6, 1959 in New York City. There will be a business meeting for members at 10:00 A.M. This will be followed by a luncheon and scientific session for members and friends starting at 12:30 P.M. The topic of the afternoon scientific session is entitled "Restoring the Occlusion." This symposium will be discussed from three different points of view: Periodontia, Orthodontia and Oral Rehabilitation. Participating in the program will be:

1. Dr. Sigurd Ramfjord, Chairman of the Periodontia Department of the University of Michigan School of Dentistry, Ann Arbor, Michigan.
2. Dr. Herbert I. Margolis, Chairman of the Orthodontia Department, Tufts University School of Dental Medicine, Boston, Mass.
3. Dr. I. Franklin Miller, Member of the American Denture Society and Fellow of the New York Academy of Prosthodontics and of the International College of Dentists.

All members of the Academy and interested dentists and physicians are cordially invited to attend. For reservations and programs address the Chairman, Dr. Andrew Cannistraci, 2152 Muliner Ave., New York 62, New York.

NEW JERSEY SECTION

Carlton Fredericks, Ph.D., radio commentator, nutrition research consultant and faculty member of Fairleigh Dickinson Dental School,, will be the guest speaker at the October 13, 1959, meeting of the New Jersey Section of the American Academy of Dental Medicine. His topic will be "Nutrition and Dental Health." The dinner meeting will begin at 7:00 p.m. at the Hotel Douglas, Newark, New Jersey.

Wives, dental assistants, and hygienists are invited to attend both the dinner and the meeting. The price will be $4 for Academy members and $5 for guests. For reservations, contact Dr. James B. Dickinson, 95 High Street, East Rutherford, N. J.

LOUIS B. KELSTEN, *Program Chairman*

Plan Now To Attend

THE FOURTEENTH ANNUAL MEETING OF THE AMERICAN ACADEMY OF DENTAL MEDICINE

Philadelphia, Pennsylvania

OVER THE MEMORIAL DAY WEEKEND

May 27th-30th, 1960

FEE SCHEDULE FOR 14th MID-ANNUAL MEETING

Hotel Sheraton-McAlpin, New York, N. Y., Sunday, Dec. 6, 1959

Members:	Luncheon and Scientific Session	$10.00
	Scientific session only	$ 3.00
Non-Members:	Luncheon and Scientific Session	$10.00
	Scientific session only	$ 5.00

DR. RUDOLPH PALERMO
111 Larch Drive
New Hyde Park, L. I., N. Y.

DEAR DR. PALERMO:

I will attend the WINTER MEETING of the AMERICAN ACADEMY OF DENTAL MEDICINE on SUNDAY, DECEMBER 6, 1959, at the Sheraton-McAlpin Hotel in New York City.

☐ Enclosed is my check for $10.00 for the Luncheon.

DR. .....
(Please Print)

Address .....

THIRTEENTH ANNUAL MEETING CLINICIANS

Emanuel Cheraskin, M.D., D.M.D. | Lester R. Cahn, D.D.S., F.D.S.R.C.S. | S. Sigmund Stahl, D.D.S., M.S.

The Thirteenth Annual Meeting of the American Academy of Dental Medicine which was held at the Hotel Claridge, Atlantic City May 14th to 17th 1959, will long be remembered as one of the finest Annual Meetings in our history. It is a rare occasion when excellent clinicians, delicious cuisine, and warm hospitality are all present in quality and abundance. This is a tribute to Drs. Reuben Feltman and Frank Schambach, General Chairmen, ably assisted by their hard working Convention Committee.

The theme of the convention during the first two days was entitled "The Role of Oral Medicine in the Practice of Dentistry". Dr. Emanuel Cheraskin, Professor and Chairman, Section on Oral Medicine of the University of Alabama School of Dentistry lectured on "The Basis for Roentgenographic Manifestations of Oral Disease". He was followed by another able speaker, Dr. Lester R. Cahn, Associate Professor, Chairman of the Division of Oral Pathology, Columbia University School of Dental and Oral Surgery, who spoke on "Ulcerous and Granulamatous Lesions of the Mouth". Dr. S. Sigmund Stahl, Assistant Professor, Department of Periodontia and Oral Medicine, New York University College of Dentistry, gave a fine presentation on "The Interrelation of Systemic Imbalances and the Behavior of Oral Tissues". This presentation was followed by one entitled "The Oral Microbiota in Health and Disease" which was presented by Dr. Harry Blechman, Associate Professor and Chairman, Department of Microbiology, New York University College of Dentistry.

Dr. Austin H. Kutscher, Research Associate and Lecturer in Clinical Pharmacotherapeutics, School of Dental and Oral Surgery, Columbia University, discussed "Drug Therapy of Oral Diseases-1958". Dr. Harold J. Jeghers, Professor of Medicine, Seton Hall College of Medicine and Dentistry, discussed "Oral Lesions Pathognomonic of Systemic Disease". His presentation was well received.

The third day's scientific session was devoted to "Endodontics". Dr. Bernard W. Greenberg, Assistant Professor and Chairman of the Department of Endodontics, Fairleigh Dickinson University School of Dentistry, was the moderator. "Rationale for Successful Endodontics" was presented by Dr. Morris B. Auerbach, Associate Professor and Chairman, Department of Endodontics, New York University College of Dentistry. The program was terminated by Dr. George G. Stewart, Assistant Professor of Endodontics, Temple University School of Dentistry, who lectured on "The Relationship of Endodontics to Oral Medicine".

Credit for this excellent scientific program goes to Dr. Abram I. Chasens, who devoted a great deal of time and effort to present these excellent panalists.

Harry Blechman, D.D.S., M.A.

Austin H. Kutscher, D.D.S.

Harold J. Jeghers, M.D.

Bernard W. Greenberg, D.D.S.

Morris B. Auerbach, D.D.S.

George G. Stewart, D.D.S.

DOCTORS HAMILTON B. G. ROBINSON AND PAUL E. BOYLE ELECTED TO HONORARY MEMBERSHIP IN THE AMERICAN ACADEMY OF DENTAL MEDICINE

Dr. Don C. Lyons, President of the American Academy of Dental Medicine presents an honorary membership plaque to Dr. Hamilton B. G. Robinson.

Dr. Robinson was awarded Honorary Membership for his contributions to dentistry, dental education and dental literature. A graduate of the University of Pennsylvania School of Dentistry, Dr. Robinson obtained his M.S. in Pathology at the University of Rochester. At present he is Dean and Professor of Oral Pathology and Diagnosis at the University of Kansas City. He is consultant to the Kansas City Veterans Administration Hospital, as well as several other hospitals in the Kansas City area. Dr. Robinson is a member of Omicron Kappa Upsilon, Sigma Xi and F.I.C.D. (Hon).

Dr. Paul E. Boyle is recipient of an honorary membership in the American Academy of Dental Medicine. He was presented the membership plaque by Dr. Don C. Lyons.

Dr. Boyle's accomplishments in the field of dental medicine are many. He was graduated from Harvard Dental School, and devoted his early years to private practice and teaching at his Alma Mater. At present he is Dean and Professor of Dental Medicine at the School of Dentistry, Western Reserve University. Dr. Boyle is Consultant and Member of the Edward Reiter Cleft Palate Clinic at Mount Sinai Hospital in Cleveland. He is a member of Sigma XI, Omicron Kappa Upsilon and Psi Omega. Dr. Boyle has contributed an amazing amount of material to dental literature, for both periodicals and textbooks.

DR. ALFONSO LENG H., OF SANTIAGO, CHILE AWARDED HONORARY MEMBERSHIP IN THE ACADEMY

Dr. Alfonso Leng H., prominent Chilean dentist, was elected to Honorary membership in absentia, at the Thirteenth Annual Meeting of the American Academy of Dental Medicine last May. Dr. Leng graduated from the dental school in Santiago, Chile in 1910 and taught Periodontology there from 1924 to 1947. He was president of the Chilean Academy of Periodontology and a member of periodontia groups in Peru and Cuba. During his career Dr. Leng served for six years as Dean of the Dental College. For almost forty years he has devoted himself to the teaching of periodontology and elevating the standards of dentistry, not only in his country but throughout South America.

FELLOWSHIPS IN THE AMERICAN ACADEMY OF DENTAL MEDICINE AWARDED TO DOCTORS BURKET, ARCHAMBAULT, WITKIN, AND YUDKOFF

Dr. Lester W. Burket, (left) receives his Fellowship Certificate from Drs. Don C. Lyons and Harold R. Gelhaar. The presentation was made at the thirteenth Annual Meeting of the Academy last May in Atlantic City. At present Dr. Burket is Dean of the School of Dentistry of the University of Pennsylvania. He is the author of several textbooks on dentistry, including the widely read book entitled "Oral Medicine."

Dr. Marcel B. Archambault, (left) is presented with his Fellowship Certificate by Drs. Don C. Lyons and George F. Clarke. Dr. Archambault, our first Canadian President, practices dentistry in Montreal, and has been very active in Academy affairs.

Dr. George J. Witkin, (left) is recipient of the Fellowship Award from two smiling Past Presidents, Dr. Don C. Lyons and Dr. J. Lewis Blass. Dr. Witkin is Associate Professor of Periodontia and Oral Medicine at New York University College of Dentistry and limits his private practice to Periodontia.

Dr. Irving Yudkoff, (left) is awarded the Fellowship Certificate by Dr. Don C. Lyons. The presentation was made by Dr. S. Leonard Rosenthal. Dr. Yudkoff is Assistant Clinical Professor in Dental Surgery (Periodontology) at the Albert Einstein College of Medicine and limits his private practice to Periodontia in association with Dr. Marvin N. Okun.

Announcements

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The following postgraduate courses in Periodontia and Oral Medicine will be offered at the New York University College of Dentistry and will be given by Dr. Sidney Sorrin and associates:

No. 124.02. REFRESHER COURSE IN PERIODONTIA AND ORAL MEDICINE

Ten sessions, Wednesday, 4:00 to 6:00 P.M. February 3, 1960 to April 6, 1960.
Tutition and books. $156.00. Instruments approximately $55.00.

Theoretical and practical application of periodontal diagnosis and techniques to general dentistry. Four lecture and six clinical demonstration sessions. Equilibration of occulsion is performed by the student on casts. Patient demonstrations include equilibration of occulsion, conservative and radical therapy, home care, etc.

No. 123.02. PERIODONTIA AND ORAL MEDICINE IN THE SPANISH LANGUAGE

Four weeks, Monday through Friday, 9:00 A.M. to 4:00 P.M.
March 7 to April 1, 1960 inclusive. Tuition including books and instruments approximately $385.00.

Theoretical and practical application of Periodontia and Oral Medicine is fully explained and demonstrated. Equilibration of occlusion is performed by the student on casts. Lectures and clinical demonstrations of diagnosis and treatment planning, conservative and radical techniques, equilibration of occlusion, home care, etc. The entire course is conducted in the Spanish language.

COMPREHENSIVE PREPARATION IN PERIODONTIA AND ORAL MEDICINE

No. 121.11 One academic year, full time. September 14, 1959 to May 27, 1960.
Tuition $1,010.00. Instruments and books approximately $135.-00.

No. 120.11 and 121.11. Two academic years, full time. September 14, 1959 to May 26, 1961.
Tuition $2,020.00 (two years). Instruments and books approximately $231.00.

No. 121.21. Two academic years, half time. September 14, 1959 to May 26, 1961. (Two and one half days weekly.)
Tuition $1,010.00 (two years). Instruments and books approximately $135.00.

No. 120.21 & 121.21 Four academic years, half time. September 14, 1959, to May 1963. (Two and one half days weekly.)
Tuition $2,020.00 (four years). Instruments and books approximately $231.00.

These courses are designed for comprehensive preparation in Periodontia and Oral Medicine and are of particular significance for certification towards specialization. All didactic and clinical phases are covered with the objective of developing expert proficiency.

No. 222. GRADUATE COURSE IN PERIODONTIA AND ORAL MEDICINE.

Two academic years, full or half time, beginning Sept. 14, 1959. Tuition $2,020.00. (two years). Instruments and books approximately $275.00.

This program leads to the degree of Master of Science (in Dentistry). The objectives are to teach superior proficiency in Periodontia and Oral Medicine based on adequate background in the basic sciences.

For further information, write to the Secretary, Postgraduate Studies, New York University College of Dentistry, 421 First Ave., New York, N. Y.

UNIVERSITY OF MINNESOTA

The University of Minnesota School of Dentistry is most pleased to announce the 3rd Annual Course in Advanced Oral Pathology for 1960. The course, held from Thursday, 21 April through Tuesday, 26 April 1960, devotes about half time to examination of approximately 200 slides.

The course will be limited to 20 participants. Tuition will be $100.00. Interested individuals will please contact: Dr. Robert J. Gorlin, Professor and Chairman, Division of Oral Pathology, School of Dentistry, University of Minnesota, Minneapolis 14, Minnesota.

SPECIAL CLINICS ON PRACTICE PROBLEMS TO FEATURE GREATER NEW YORK DENTAL MEETING

A special, all-day series of clinics on practice problems by leading specialists of the New York area will feature the 35th annual Greater New York Dental Meeting, world's largest seminar of post-graduate dental education, to be held at the Hotel Statler Hilton, New York City, December 7 to 11, it has been announced by Dr. Adolph G. Wagner, General Chairman.

The meeting program, Dr. Wagner emphasized, will avoid duplication of clinicians appearing on the scientific program of the American Dental Association's Centennial session.

Newer phases of mouth rehabilitation will be stressed in a post-graduate study course. In addition, a number of innovations will be introduced for the first time, as well as many features that proved particularly popular at the 1958 meeting.

The program will comprise joint presentations of new research developments by leading investigators of dental schools, discussions of uses and abuses of antibiotics and tranquilizers, the ladies' luncheon and fashion show, and the "Luncheon and Learning" session, at which dental leaders seated at heads of tables seating ten persons will moderate informal discussions of their specialties. In addition, the registered clinics, visual education films, clinical demonstrations and other educational features that have always distinguished the meeting will be included.

In general, Dr. Wagner reported, the 1959 session will present the latest current procedures in all phases of dentistry, as well as studies of new researches significant to future dental practice. All presentations listed in the printed program will carry expanded synopses of that subject matter so that registrants may have in advance a clear grasp of their scope and substance.

For programs and reservations, please write Mrs. Mabel Purdy, Executive Secretary, Room 106A, Hotel Statler Hilton, New York 1, N. Y.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

DPG. 701—A COMPREHENSIVE COURSE IN COMPLETE DENTURE CONSTRUCTION

Each Wednesday on the following dates:
October 28, November 4, 18, 25, 1959
Tuition $125.00—Class Limited
Dr. Irving R. Hardy, Professor of Prosthetic Dentistry (Emeritus 1958)

For further information and application write to: Director of Graduate and Postgraduate Studies, 136 Harrison Avenue, Boston, Massachusetts.

UNIVERSITY OF PITTSBURGH SCHOOL OF DENTISTRY

The University of Pittsburgh and the Odontological Society of Western Pennsylvania are sponsoring several postgraduate courses in the Fall and Winter of this year.

Oral Diagnosis-Treatment Planning
Dr. Lewis Fox, October 21, 22, 23, 1959.
Tuition $150.

Full Denture Construction
Dr. Ernest T. Lewis, November 11, 12, 13, 14, 1959.
Tuition $200.

Modern Approach to Operative Dentistry
Dr. Henry M. Tanner, Feb. 11, 12, 13, 1960.
Tuition $100.

For further information and an application blank, contact The Odontological Society of Western Pennsylvania, 206 Jenkins Arcade, Pittsburgh 22, Pennsylvania.

SOCIETY OF ORAL PHYSIOLOGY AND OCCLUSION

The Sixth Annual Meeting of the Society of Oral Physiology and Occlusion will take place on Saturday, December 5, 1959 in the Empire Room at the Hotel New Yorker, 34th St. and 8th Av., New York City. The seminar will be scheduled from 9 A.M. to 5 P.M. and the subject will be "Pain and the Temporo-Mandibular Joint." Among the speakers will be Dr. Janet Travell, Dr. Joseph J. Martini and Dr. Clyde B. Kyne. Those interested in joining this organization should contact Dr. S. M. Kelner, Sec'y, 57 West 57th St., New York, N. Y.

DENTAL GUIDANCE COUNCIL FOR CEREBRAL PALSY

The Annual Scientific Meeting of the Dental Guidance Council for Cerebral Palsy will be held in the Washington Room of the Hotel Statler, 32nd Street and Seventh Avenue, New York City on Thursday evening, November 12, 1959 at 8 P.M.

This symposium should be of particular interest to the dental profession and all members and their friends are cordially invited to attend. Further information may be secured at the offices of the Dental Guidance Council for Cerebral Palsy, 70 Fifth Avenue, New York 11, New York.

AMERICAN CANCER SOCIETY, INC.

A "Symposium on Evaluation of Early Diagnosis of Cancer" to be presented at the Annual Scientific Session of the American Cancer Society to be held October 26-27, 1959 at the Biltmore Hotel, New York, has recently been announced.

This program is fully approved and recommended

by the American Academy of General Practice for 12 hours of Category II Credit for its members.

For further information, contact Dr. Scott Hill, Director, Professional Education, American Cancer Society, Inc., 521 West 57th Street, New York 19, N. Y.

ST. LOUIS DENTAL SOCIETY

The 1959 Mid-Continent Dental Congress will feature one of the most unusual dental programs ever presented. The entire program will be given by the Indianapolis Dental Research Group under the direction of Dr. John Geller. This program, directed to the general practitioner, has been two years in preparation and its aim is to present material which can be taken back to the individual office and used. The Congress will be held November 1-4, 1959 at the Chase Park Plaza Hotel in St. Louis.

All A.D.A. members are cordially invited to attend the Mid-Continent Dental Congress. For further information contact James E. Brophy, Executive Secretary, St. Louis Dental Society, 8013 Maryland Avenue, St. Louis 5, Missouri.

ORAL HYGIENE COMMITTEE OF GREATER NEW YORK

Title: Dental Health Education in Relation to Practice Management
Sponsor: Oral Hygiene Committee of Greater New York
Date: Sunday, October 18, 1959
Time: 10:00 A.M. to 1:00 P.M.
Place: Second District Dental Society, 111 Ft. Greene Place, Brooklyn 17, N. Y.

SPEAKERS

1. Dr. FRANCES A. STOLL, Professor of Dental Hygiene, Columbia University, School of Dentistry

She will present techniques for motivation of interest in Dental Health among school children. It will include a discussion of the role of the dentist and hygienist in the education of school children and in their motivation to greater appreciation of dental health and their role in maintaining it.

2. Dr. J. LEWIS BLASS, Professor of Periodontia and Oral Medicine, New York University, College of Dentistry

He will present practical applications of Dental Health Education for the dentist to use with patients. These are intended to provide more personal satisfaction in the practice of Dentistry, and create greater cooperation on the part of the patient and should lead to greater financial rewards.

The dentist's application of the techniques of the two speakers will enable him to follow through on methods used in the community and the school and obtain better results from his own efforts in the education of patients.

The basic aim of the day's work will be to explore means for getting the patient to accept more of the dentist's services and to gain his cooperation in achieving and maintaining dental health.

UNIVERSITY OF ALABAMA

The University of Alabama School of Dentistry announces the following refresher courses which will be presented during the months of October, November, December, and January:

October 3, 4, 1959: *Oral Rehabilitation,* Jerome M. Schweitzer, D.D.S.

October 10, 11, 12, 1959: *Hypnosis Applied to Dentistry,* William T. Heron, A.B., M.A., Ph.D.

October 17, 18, 1959: *Insurance, Investments & Estate Planning,* A. Key Foster, A.B., LL.B.

October 24, 25, 26, 1959: *Root Canal Therapy,* Adeeb E. Thomas, D.M.D.

October 30, 31, November 1, 1959: *Crown & Bridge Prosthodontics* with emphasis on pontic construction and fused porcelain veneers, John F. Johnston, D.D.S.

November 7, 1959: *Hospital Protocol for the General Practitioner,* Charles A. McGallum, D.M.D., M.D.

November 14, 15, 1959: *Oral Cancer,* Leonard Robinson, D.M.D., M.D.

November 21, 22, 23, 1959: *Major Oral Surgery,* Robert B. Shira, Col., DC. USA (Limited to oral surgeons)

December 5, 6, 7, 1959: *Preventive Orthodontics,* Sidney B. Finn, D.M.D., M.S.

January 16, 17, 18, 1960: *Clinical & Laboratory Procedures in Crown and Bridge,* William D. Powell, Jr., D.M.D.

January 22, 23, 24, 1960: *Oral Diagnosis, Roentgenographic Interpretation, & Treatment Planning*: A Correlation with Oral Pathology, Donald T. Waggener, D.D.S.

Further information regarding these courses may be obtained by writing The Director, Refresher Course Program, University of Alabama School of Dentistry, 1919 7th Avenue South, Birmingham, Alabama.

TEMPLE UNIVERSITY SCHOOL OF DENTISTRY GRADUATE AND POST GRADUATE PROGRAM FOR 1959-60

Clinical Endodontics

Leonard Parris, D.D.S. and Associates
October 26, 27, 28, 29, 30, 31, 1959
9 A.M. to 5 P.M. daily

Clinical Oral Pathology
Max Jacobs, D.M.D., M.D.
January 14, 15, 16, 1960
9 A.M. to 5 P.M. daily

Advanced Orthodontics
Robert H. W. Strang, M.D., D.D.S., and Associates Will M. Thompson, D.D.S. and Glenn H. Whitson, D.D.S.
January 17 to 30, 1960

Anatomy—Head and Neck Dissection
Richard M. Snodgrasse, Ph.B., M.A., Ph.D.
February 3 to June 8, 1960—Fifteen Wednesdays from 1 to 5 P.M.

Advanced Fixed Prosthesis
George Straussberg, D.D.S.
February 8, 9, 10, 11, 12, 13, 1960
9 A.M. to 5 P.M. daily

Periodontal Prosthesis
Morton Amsterdam, D.D.S., F.A.C.D. and D. Walter Cohen, D.D.S.
February 8, 9, 10, 11, 12, 1960
9 A.M. to 5 P.M. daily

Present Day Periodontal Practice
Jacoby T. Rothner, D.D.S., F.A.C.D., Bernard B. Saturen, D.D.S., and Neal W. Chilton, B.S., D.D.S., M.P.H.
One day a week for 10 weeks Starting Tuesday, February 16 through Tuesday, April 26, 1960.

Practice Administration
Jay H. Eshleman, D.D.S., D.Sc.
February 27, 1960 9 AM. to 5 P.M.

Clinical Diagnosis and Oral Medicine
Lester W. Burket, D.D.S., M.D., D.Sc., F.A.D.M. and S. Leonard Rosenthal, D.D.S., F.A.C.D., F.A.D.M.
February 29 to March 4, 1960
9 A.M. to 5 P.M. daily

Electronic Electrosurgery
Maurice J. Oringer, D.D.S.
March 18, 19, 1960
9 A.M. to 5 P.M. daily

Full Denture Prosthesis
Bernard Jankelson, D.M.D. and Associates
March 21, 22, 23, 24, 25, 26, 1960
9 A.M. to 5 P.M. daily

Advanced Surgical Techniques in Periodontal Therapy
Saul Schluger, D.D.S. and Jack Alloy, D.D.S.
April 4, 5, 6, 7, 8, 9, 1960
9 A.M. to 5 P.M. daily

Graduate Orthodontics
Robert B. Hedges, D.D.S., M.S., F.A.C.D., and Associates
September 12, 1960—A two year course
For application and additional information, write to: Dr. Louis Herman, Director of Postgraduate Studies, Temple University School of Dentistry, 3223 North Broad Street, Philadelphia 40, Pennsylvania.

NORTHEASTERN ASSOCIATION OF DENTAL EDITORS

The third Annual Dental Writing Conference of the Northeastern Association of Dental Editors, will be held on December 2nd 1959 at the New York Academy of Science, 2 East 63rd Street, New York City.

As an eagerly awaited annual event, these conferences have evoked great interest among dental editors and all dentists interested in writing. Once again this year's conference will be supported by a grant from the William J. Gies Foundation for the Advancement of Dentistry.

This year's conference will deal with the resources and source materials available to the dental editor and dental scientific or editorial writer, in the field of health, in order to help him distinguish fact from fiction. Guarding the accuracy of a publication or scientific paper, and improving its content as the main responsibility of the dental writer, will be explored by a group of eminent experts.

The President of the Northeastern Association of Dental Editors, Dr. Joseph H. Kauffman, has extended a cordial invitation to previous participants and especially to all other interested members of the profession to attend.

Further information may be obtained by addressing the chairman of the Writing Conference of the NEADE, Dr. Paul Scheman, 134 West 58th Street, New York 19, N. Y.

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